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NEW ENGLAND PEDIATRIC SOCIETY

A meeting of the Society was called to order by the President, Dr. Lewis Webb Hill, Boston, at 8:15 P. M., on May 6, 1932 who spoke as follows:

This meeting represents an attempt to arrive at conclusions concerning the rational use of the vitamin preparations in pediatric practice. There is one man whose work on deficiency diseases and allied subjects has been so brilliant and so applicable to the everyday work of each one of us that any such meeting as this could not be complete without his presence—Dr. Alfred Hess of New York.

DIET, NUTRITION AND INFECTION*

BY ALFRED F. HESS, M.D.†

IT is a commonplace that the relationship is intimate between composition of the diet and susceptibility to infection. However, the extent of this relationship and its importance in clinical medicine has only just begun to be realized; in fact we are still uncertain as to the limits of altered susceptibility. From the standpoint of disease, diet, nutrition and resistance to infection should be regarded as an etiologic unit rather than as a triad. In appraising dietaries from this point of view, not only the several vitamins should be considered, but the various inorganic and organic constituents which likewise may be implicated in bacterial infection. It would lead too far afield, however, to consider these various aspects of the subject, so that I shall confine myself to the rôle of some of the vitamins, basing my conclusions mainly on observations made during the past ten to fifteen years in a child-caring institution. As my experience has been concerned chiefly with the antirachitic, antiophthalmic and antiscorbutic vitamins, in other words with vitamins D, A and C, I shall limit my comments to these specific nutritional factors. Furthermore, I shall take into consideration only clinical data, to the exclusion of experiments on animals.

After an experience of several years with the effect of *ultraviolet rays* in the prevention and cure of rickets, an effort was made to lessen the incidence of infection in the institution by means of irradiation with the mercury vapor lamp. As is well-known, respiratory infections constitute one of the last vestiges of institutionalism in hospitals and asylums for children and, during the winter months, plague and torment their foster-parents. Our first attempt, undertaken in 1926[1] with the confidence born of inexperience, was most disappointing. In the course of the winter, in spite of irradiation carried out every other day for a period embracing four months, quite as many infections occurred among the group of infants who were irradiated as among those who lived under the same régime except that they were not irradiated. It may be added that the irradiated group evidenced an initial increase in weight which, however, did not continue during the subsequent months.

Two years later a similar investigation was carried out[2] with the only difference that a carbon arc lamp was used as the source of radiation, as it was thought that these rays might be superior because they more nearly resemble the spectrum of the sun. Again our efforts were fruitless. In spite of systematic exposures to these rays no relative diminution in the incidence of respiratory infections occurred during an observational period of three months.

The following year, 1929, the problem of infection was attacked in a different way[3]. Rickets was prevented by means of the usual doses of cod liver oil, in other words of three teaspoonfuls daily for babies three months or more of age. The diet was composed of full amounts of pasteurized milk, cereals, orange juice, and of vegetables for the older infants. In order to render exposure as infrequent as possible, what was termed "aseptic nursing" was carried out in one ward—physicians, nurses and attendants coming in contact with the infants were required to wear surgical masks which were changed daily; hands were scrubbed thoroughly and frequently; visiting was allowed but once a month and visitors were provided with masks; fondling and petting of infants were prohibited and nurses who had colds or infections were temporarily excluded from service. Once again our attempts at prophylaxis resulted in failure; infections

*Read before the New England Pediatric Society at its meeting, May 6, 1932.

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were frequent in spite of all hygienic precautions and the inclusion of cod liver oil in a dietary which already was liberal. A graph (Chart 1) is reproduced to illustrate the irregularity and uncertainty in the occurrence of infections rather than to compare their incidence among the children in the "aseptic ward" and those in an ordinary ward. It shows that among the latter there was an epidemic of respiratory infections during the month of November, whereas among the former there was a violent outbreak in March. The diet of both groups was not only the same, but unchanged throughout the period. The occurrence of "grippe" was not due to dietary deficiency but to some fortuitous occurrence, probably to the chance introduction of a highly infective agent.

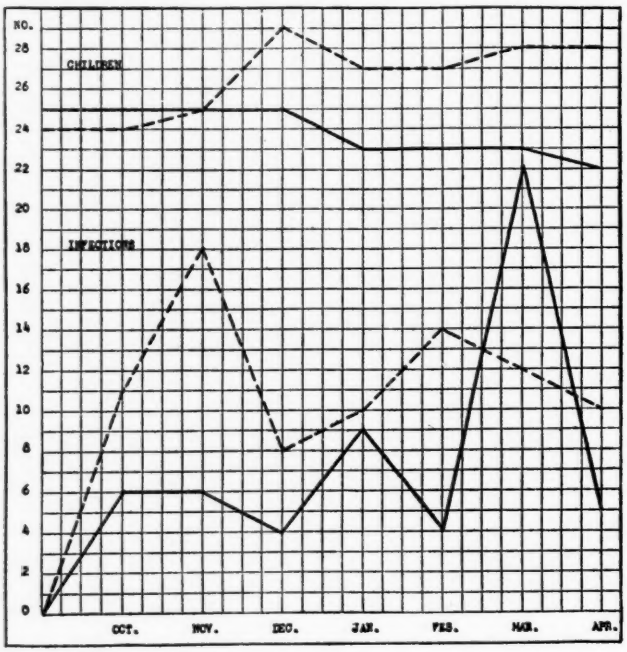

CHART 1. Outbreak of Infection in protected ward (broken line) in November. Outbreak in control ward (solid line) in March.

These and similar experiences have led, in the first place, to the conclusion that mild rickets does not predispose to infection and that anti-rachitics—ultraviolet irradiation or cod liver oil—do not lessen their incidence. Severe rickets probably does entail increased susceptibility, but the rickets to which we have reference is the prevalent type throughout the community that is manifested by beading of the ribs, occasional craniotabes, diminution of the inorganic phosphorus in the blood and, in about one-third of the cases, slight roentgenologic changes at the epiphyses. Not only is there no disposition to infection in this type of rachitic child, but infections have shown no tendency to be unduly severe. In a tabulation of the incidence of pneumonia of 114 mildly rachitic infants and of 102 non-rachitic infants, who had been protected by means of cod liver oil or ultraviolet irradiation, the superiority happened to be with the mildly rachitic group which did not receive oil.

What was found to be true of mild rickets held likewise for general nutrition, as judged by weight; this factor bore no relationship to the occurrence or to the fatality of pneumonia, with the possible exception of infants under six months of age.

Of late, *vitamin A* has been accorded the greatest credit in warding off infection, and a deficiency of this factor in increasing susceptibility. In fact vitamin A is at times termed the "anti-infectious vitamin". This designation has tended to replace the term "growth vitamin" since it has been realized that neither this nutritional factor nor any other has been entrusted with the control of the growth of the body. All who have carried out investigations in this field have found that growth is affected adversely by a deficiency of any one of many food constituents, for example, lack of an inorganic salt. However, the rôle of this vitamin in protecting the body against infection, more especially against invasions of the respiratory tract, has gained an increasingly strong foothold. Since the classic publication of Bloch[4] in 1917 there can be no doubt that a lack of vitamin A may induce a heightened susceptibility to respiratory infections. But, it should be borne in mind that the infants that formed the basis of Bloch's report suffered from malnutrition and had been subjected to a marked deficiency of this vitamin. For us, the important question is whether the ordinary diet of infants, children and adults, includes an

adequate quantity of this nutritional factor and, in turn, whether under prevailing dietary conditions, a deficiency of this vitamin can be held accountable for the high incidence of respiratory infections throughout the community. Although it may be stated at the outset that it is not possible at present to give a categorical answer to this question, there are sufficient clinical data to render a discussion of this important question quite worthwhile.

In the first place let me call to mind again the experience of 1929, in which the addition of cod liver oil to a liberal dietary failed to prevent explosive outbreaks of "grippe" in November and in March. During the winter of 1930-31 a very painstaking investigation of this problem which was carried out in my clinic by Barenberg and Lewis[5], led to similar failure. Without reciting the details of this study, which will be found in the original communication, it may be stated that a large series of infants was given a diet especially rich in vitamin A—including not only milk, but butter as well as vegetables, and that in the course of an observational period varying from four months to a year, all of these infants developed one or more infections. Furthermore, it proved to be immaterial whether the infants received still greater amounts of this vitamin, for respiratory infections were not diminished in number or in severity among a large group which was given the customary amounts of cod liver oil, nor even among a smaller group which received double the usual quantity.

Our experience during the past winter has served only to strengthen the impression gained during previous years. In the course of a test of the antirachitic potency of several dietaries, one series of infants was given a certified raw milk which was assayed and found to be rich in vitamin A, another series the same kind of milk which had been enhanced in its vitamin D content by feeding the cows irradiated yeast ("vitamin D milk") and a third series was fed pasteurized milk and in addition received three teaspoonfuls of cod liver oil daily. The other constituents of the diet were much the same as outlined in connection with the test of 1930-31. Although the investigation was undertaken primarily in connection with rickets, it serves as a gauge of the efficacy of vitamin A as "the anti-infectious vitamin". In regard to the incidence of infection, the results of this study were similar to those of former years. In other words, no difference was noted in the number or in the severity of respiratory infections among the members of the different groups, notwithstanding the fact that one group was given the benefit of a large addition of vitamin A by supplementing the dietary with cod liver oil. These infants were about three to six months of age at the outset and were under close observation for a half year or more. It is not so much that no distinction, in regard to infection, could be drawn between the members of these groups, but that the absolute number of infections was unusually high in all three. By infections I have in mind respiratory disturbances which are manifested by catarrhal symptoms and a rise in temperature to 100° F. or above. Indeed, in spite of the liberal diet and of the fact that they were cared for in individual cubicles and that the hygienic surroundings were unexcelled, every infant developed almost one infection a month. Major respiratory diseases were few in number; but whether or not pneumonia, empyema, otitis media or mastoid disease develops is merely a matter of chance, depending on the nature of the infective agent and, probably, on other factors with which we are as yet insufficiently informed. That such is the case we have been forced to conclude as the result of a prolonged experience, during the course of which we have at times been led to believe, perhaps for a period of five years or more, that we had gained mastery over severe infections, only to be disillusioned by a subsequent year*.

In connection with this consideration of the value of vitamin A in protecting infants against respiratory disorders, it seemed worthwhile to consult the records of a group of children which manifested "carotinemia" some years ago. This name was given in 1919, by Hess and Myers[6], to a clinical condition in which the skin became yellow and carotin was found in high concentration in the blood; it came about as the result of feeding a diet which contained a large amount of carrots and spinach. As has been recently shown, by v. Euler and others, carotin must be regarded as the "provitamin" of vitamin A, capable of transformation into the specific vitamin within the animal body. It is true that this chemical metamorphosis has not been definitely proved in regard to man. On consulting the histories of these cases it was found that infections were by no means infrequent during this period, in fact that in some cases "grippe" had been noted every few weeks. If carotin can be transmuted into vitamin A, these infants must have been thoroughly saturated with this nutritional factor. In this connection it should be mentioned that carotinemia is found most commonly in persons suffering from diabetes, due to their consumption of exceptionally large amounts of vegetables rich in carotin. Nevertheless, as is generally accepted, diabetics are prone rather than immune to infection.

*It may be added, that we have attempted to cure vaginitis, gingivitis and stomatitis, infections which involve ectodermal tissues, by means of feeding large amounts of vitamin in the form of cod liver oil, but have not found this therapy of benefit. Furthermore, infectious diseases such as measles, when occasionally they have broken through quarantine and gained access to the wards, attacked the children indiscriminately, quite independent of whether they were getting a diet enriched with cod liver oil. Measles is singled out because it must be regarded as a respiratory disease from a pathogenetic point of view. The same indiscriminate distribution is true of chickenpox, which is still more infectious and can only occasionally be limited by isolating infants in cubicles. More infectious, in our experience, than either of these highly communicable disorders is the common "grippe" of winter and early spring.

In the course of these various studies it has become increasingly evident that during infancy, even during the first year of life, *age* plays an important rôle in determining whether infection comes about. That such is the case was clearly brought out some years ago in a tabulation which showed, for a period of ten years, the incidence of pneumonia in the institution in relation to the age distribution of the children. As will be seen (Table 1), among some

TABLE 1

AGE DISTRIBUTION OF CHILDREN IN INSTITUTION (1916-1927) AND OF THOSE WITH PNEUMONIA

Age	Population, 1916-1927	Per Cent of Total	Cases of Pneumonia, 1916-1927	Case Rate per Age Group. Per Cent
0 to 6 months	505	7.3	22	4.3
6 months to 1 year	739	10.7	106	13.0
1 year to 2 years	1,392	20.2	214	15.4
2 to 3 years	1,318	19.1	105	8.0
3 to 4 years	1,317	19.1	41	3.1
4 to 5 years	1,623	23.6	17	1.0
Totals	6,894	100.0	505	7.3

1,200 infants under one year of age, the incidence was three times as great in those six to twelve months as in those under six months of age. The fatality rate, however, was higher in the younger group. Curiously enough the importance of the factor of age was lost sight of until last year when it was again brought to our attention by a striking incident. For many years we had made use of a cubicle system for quarantining infants during the first few weeks of their stay in the institution. On account of the good results obtained with this method, a large cubicle system was recently constructed for the permanent care of infants. On the first floor those under six months were placed, and on the second floor those about six to twelve months of age. It became increasingly evident that the number of infections among the younger infants on the ground floor was decidedly fewer than among those on the second floor. The usual explanations were thought of, such as defects in nursing technic or some unknown carrier of infection. In order to gain additional information in regard to this divergence, a few of the younger infants were transferred to the second floor and a few of the older ones to the ground floor. The result of this change demonstrated that the difference in incidence of infections did not depend on the character of the nursing or the situation of the cubicles but was inherent in the age of the infants—those under six months of age being definitely less subject to respiratory infections than those between six and twelve months. I emphasize this clinical phenomenon in the first place, because it has not been given significance in computing morbidity statistics in institutions or in communities. It indicates that, during the first months of life, in regard to immunity from infection, the infant is still leading a somewhat parasitic existence, depending largely on the protective substances carried over from the mother. I wish to emphasize especially in this connection, however, that this immunity cannot be attributed to vitamin A. Just as infants do not come into the world with a large store of the antirachitic factor, as a biologic assay by Miss Weinstock and me showed, so it has recently been demonstrated that the store of vitamin A is insignificant at birth. Wolff[7] found that the livers of 62 per cent of newborn infants in Amsterdam contained none of this vitamin whatsoever and that those who had a store at the time of birth, contained this factor in but small amount. It is evident that the outstanding immunity of the very young infant to respiratory infections cannot be ascribed to an exceptional supply of vitamin A.

TABLE 2

NUMBER OF INFECTIONS IN RELATION TO AGE AMONG INFANTS RECEIVING COD LIVER OIL OR VIOSTEROL (1926, 1929, 1930)

Age (Mos.)	Number of Infants		Average Period of Therapy (Months)		Number of Infections		Infection per Infant per Month	
	Cod Liver Oil	Viosterol	Cod Liver Oil	Viosterol	Cod Liver Oil	Viosterol	Cod Liver Oil	Viosterol
0-3	27	47	2.0	1.7	8	7	0.14	0.08
3-6	61	75	2.4	2.5	29	21	0.19	0.11
6-9	64	88	2.6	2.8	56	56	0.33	0.22
9-12	57	78	2.7	2.8	59	58	0.38	0.26
12-15	48	65	2.7	2.5	31	33	0.24	0.20
15-18	37	43	2.5	2.1	16	19	0.17	0.21

I do not believe that the dietary which the children in my institution receive can be poor in vitamin A in view of its content of 24 to 32 ounces (750 to 1000 cc.) of milk, of vegetables and even of butter, and certainly that it cannot remain deficient in this particular after it is supplemented by liberal amounts of cod liver oil. In my opinion, the everyday infections which sometimes prove so serious have little or nothing to do with this deficiency, although there is no doubt that a decided or long-standing deficiency may bring about increased susceptibility. Spence[8] of Newcastle, who has carried out careful studies of this nutritional condition, has failed to note an increase of infections even among a group of children suffering from xerophthalmia and night-blindness. He found them subject merely to an increase in infections of the skin, such as impetigo and boils. Perhaps the diarrhea which has been reported in connection with fat-soluble A deficiency and which indeed is the outstanding symptom of

"hikan", a disorder reported by Mori[9], is another manifestation of infection of the surface of the body.

As is well known, Mellanby and Green[10] have stated recently that a lack of vitamin A in the diet is responsible for puerperal sepsis, and are of the opinion that they were able to prevent this serious condition by giving a vitamin A concentrate. I am not in a position to cite cases of this description from actual experience, but the distribution of puerperal sepsis throughout various countries of the world does not seem to bear out this contention. As is well known, the incidence of puerperal sepsis in the United States is very high compared with European countries. For example, the mortality is stated to be more than twice as high in this country as in towns in the European part of the Soviet Union, as reported in 1926, on the basis of over one-half million confinements[11]. It is difficult to square these statistics and those of a similar nature with the vitamin A intake and economic status of the peoples of Europe and of the United States*. Aykroyd[13] tells us that on the coast of Newfoundland, night-blindness, a symptom of vitamin A deficiency, occurs in women usually in association with pregnancy, but he adds that puerperal sepsis is not common in Newfoundland.

In connection with a consideration of infection it should be mentioned that *intestinal parasitism* has been noted by many observers in association with vitamin A deficiency. Gamboa[14] has referred to ascariasis, and Mori to the frequency of anchylostoma in the mother. Most observers attribute the vitamin deficiency to the intestinal infestation rather than that the parasitism has resulted from the food deficiency, which to my mind is the true sequence of events. In 1921, Pappenheimer, McCann and I[15], in the course of a study of diets deficient in vitamin A, pointed to the fact that infestation with cestodes was extremely common. Orr[16] has recently emphasized the close relationship between intestinal parasitism among African tribes and deficiencies in diet. It is quite possible that the notable frequency of intestinal parasitism in the Far East, in Porto Rico and elsewhere may be ascribable to one or more deficiencies in the diet. In this connection it may be of interest to cite the following comment of Hegner[17], "There is an interesting relation between these intestinal protozoa and the character of our diet. Meat-eating animals are not ordinarily infected with them, but vegetarians are very highly parasitized. Casein seems to be the best of the proteins. A diet consisting largely of casein soon brings about a decrease in the number of certain organisms and often leads to their total elimination."

The stumbling block in this entire question is to determine what constitutes an adequate amount of vitamin A in the dietary. In the rat a lack of this factor leads to a cessation of *growth* as soon as the body store of the vitamin has been used up. In fact, cessation of growth, together with xerophthalmia, is used as the criteria in assaying products for this vitamin. In infants, however, the relationship between growth and deficiency is not so intimate or reliable; otherwise we should have at hand a valuable means and measure for gauging the adequacy of vitamin A. In a study of the "clinical rôle of the fat-soluble vitamin", carried out with Unger[19] in 1920 in which a few infants were closely observed who had received a minimal quota of this vitamin for periods of some months, it was remarked that "their growth in length has been normal and their growth in weight slightly below normal". In fact, adding an increased amount of cereal, which contained practically none of the deficient vitamin, brought about a gain in weight. The experience of the past few years has served to strengthen my opinion that failure to gain in weight should not be attributed to lack of vitamin A. If we use growth as a criterion of adequacy of this vitamin, we must conclude that our infants are receiving sufficient, for on adding cod liver oil to the dietary we have not been able as a rule to bring about a gain. With this point in mind, we have recently reviewed a large number of weight charts, and have found more instances in which a failure to gain followed giving cod liver oil, than cases in which an increase in weight was brought about. In his recent study, Spence also emphasizes the fact that the growth of the children which developed xerophthalmia was but little inhibited and that their nutrition did not suffer. On the other hand, in Blegvad's cases, which advanced to the stage of keratomalacia, there was loss of weight for two or three months before the signs developed. Cases of keratomalacia are of no value in a consideration of the question of adequacy of the diet or of signs of early deficiency.

Without doubt infants are more susceptible than adults, which is true in respect to all the vitamins. Furthermore, it can be stated that there is unanimity that the first year of life is the period of greatest susceptibility. Thalberg[20], in his early description of cases from St. Petersburg, reported that most of his cases were breast-fed infants whose mothers had fasted for a considerable period. Among Bloch's 86 cases, 47 developed the disorder during the first year of life and 20 of these in the course of the first six months. Of Blegvad's 430 cases of keratomalacia, in other words of advanced deficiency, 368 occurred during the first year, the majority

*Macy and her coworkers[18] write as follows: "Milk from women on the average American dietary is apparently a relatively rich source of vitamin A, since 2.5 to 3 cc. of the mixed milk from a group of wet nurses in various stages of lactation were sufficient to satisfy the nutritive demands for this factor during growth and reproduction of the rat. Further evidence of the potency of breast milk is illustrated in the children's clinics, where ophthalmia is rarely, if ever, observed in suckling infants."

in the second quarter; 62 were seen during the second year of life[21]. The curve of incidence which he publishes reminds one of the age-incidence of infantile scurvy. The mortality likewise was greatest in the second quarter of the first year and comprised 90 deaths.

It is evident, therefore, that infants must be considered quite apart from older children or adults. This is true likewise from a symptomatic standpoint as the earliest sign of vitamin A deficiency differs in the two groups. In infants the earliest sign of deprivation is xerosis of the cornea, which is a stage antecedent to xerophthalmia, in older children and adults the earliest symptom is purely subjective and consists of night-blindness.

The data in relation to infants are less numerous and detailed than that for adults. In general it may be stated that no case of xerophthalmia has been reported which developed on whole milk, irrespective of quantity, nor in an infant that was receiving cod liver oil. From the point of view of vitamin A, these agents seem to represent adequacy, coupled with a factor of safety. Furthermore, all active cases seem to have responded promptly to milk or to cod liver oil. In regard to the oil the customary dose has been about three teaspoonfuls daily, in other words the amount which the children in my institution received and which failed to protect them against frequent respiratory infections. Mori noted a marked improvement as early as one-half day after giving cod liver oil. Monrad[22] writes that 10 gm. of whole milk brought about a prompt subsidence of xerosis in cases occasioned by a diet of milk which had been not only skimmed by centrifugation but doubly heated. If we can draw any conclusion from this and other experiences it would seem that an allowance of 24 ounces (750 cc.) or more of milk, which is the usual quota for our infants, should amply cover requirements. It may be added that Blegvad found that boiled milk was just as effective a curative agent as raw milk

As stated, older children and adults must be considered as a group separate from infants because they manifest night-blindness or nyctalopia. This is often the first symptom noted by the Russian peasants following the Lenten fast. It is intensified by sunlight, probably owing to the fact that the rays use up the visual purple in the background of the eye. It is likewise the first symptom of improvement in this deficiency disorder. Spence tells us that 10 cc. of cod liver oil cured the condition rapidly, doing away with night-blindness in older children within a period of from three to five days. Aykroyd remarks that it is the rule that the vision is normal 12 to 24 hours after liver or liver oil is given; the dose of the liver oil was usually about two tablespoonfuls. The rapidity in the disappearance of symptoms reminds us of the magic cures of polyneuritis in pigeons, brought about by feeding rice polishings or yeast. Aykroyd's note to the effect that the vision is kept normal by giving about an ounce of cod liver oil every four or five days throughout the summer is particularly interesting as it furnishes some measure of the demands and the utilization of this vitamin under extreme conditions*.

The significance of night-blindness in connection with our study is that this defect in vision is a rare clinical condition in this country and, therefore, it may be inferred that a lack of vitamin A in older children and in adults is exceptional. The Quarterly Index for 1931 does not refer to a single clinical paper from the United States on the subject. It should be mentioned, however, that night-blindness seems to be increasing in England, as Spence observed 17 cases in the course of a year in Newcastle. It is quite possible that if economic stress should be prolonged and should increase in this country, similar cases will be noted here. At present, however, the rarity of night-blindness must lead us to conclude that it is rare for the dietary of older children and adults to be deficient in this factor.

In passing, I should like to draw attention to a cheap and effective source of vitamin A which, with advantage, could be added to the human dietary, especially when a deficiency of this vitamin is suspected. As is well known, *alfalfa* is one of the richest sources of vitamin A, the only vegetable which contains a comparable amount being spinach. Like spinach, it is also rich in iron and other mineral salts. This legume is also used as a source of high-grade protein for animals. In regard to vitamin A, it is the equivalent of butter, one teaspoonful containing approximately the same number of units as a teaspoonful of average dairy butter. Biological assay shows it to have about one-half the potency of cod liver oil, but, it should be added, a level teaspoonful of alfalfa weighs only one-third as much as the oil. Having these qualities in mind we have recently incorporated alfalfa in the dietary of our children†. The simplest method was found to be to add one or more teaspoonfuls to the six-ounce portion of broth which they regularly obtain. This forms a palatable soup, having a taste similar to pea soup, and is liked by infants and young children. It has been incorporated also in the cereal immediately after cooking; one teaspoonful is added. It would seem that alfalfa could be introduced in the dietary with notable effect in Russia, China and the Far East where A avitaminosis is most common. It is at one and the same time a food which is inexpensive and

*He writes, interestingly, that after two men had been complaining for some days, one of the crew shot a sea gull and the sufferers ate half of its liver, uncooked, apiece. Their vision returned to normal in 24 hours.

†Edible alfalfa is in the form of a very fine flour and contains about seven times the vitamin A units of ordinary alfalfa, owing to the fact that it has been dried rapidly by a mechanical process rather than allowed to dry slowly in the sun (Russell).

contains a specific vitamin in exceptional concentration. It is sad to contemplate that this source of vitamin A could well have been at the disposal of Denmark during and immediately after the War period and could have averted the many cases of blindness which resulted from a deficiency of this specific factor. A similar comment is pertinent in regard to the bone disorders which occurred in Germany, Austria and Poland during the War which could have been prevented had the specific properties of solar and artificial ultraviolet rays been appreciated at that time.

In regard to *vitamin C*, I shall refer only to the bearing of this vitamin on infections, more particularly of the respiratory tract. In 1917, in a paper on the pathogenesis of infantile scurvy, I emphasized the fact that a lack of the antiscorbutic factor which leads to scurvy, at the same time predisposes to infections[23]. This enhanced susceptibility has been confirmed by Abels, Ludwig Meyer and many others. It exists even before the scorbutic signs are manifest in the stage which is better termed "latent scurvy" than "Praeskorbutus" as the abnormal scorbutic state already exists. Similar susceptibility to infections goes hand in hand with adult scurvy. This was pointed out years ago by Lind and others in connection with scurvy in the mercantile marine and among the soldiers in times of war, for example, in our Civil War and in the Crimean War.

But I wish to emphasize quite another aspect of infection in connection with infantile scurvy. In 1917, and again in 1920, I called attention to the "widespread occurrence of nasal diphtheria in infantile scurvy", remarking that "we have encountered nasal diphtheria—with typical bloody mucus discharge—so frequently in connection with scurvy that where this local infection occurs among a group of infants they should be carefully examined for latent or mild scurvy"[24]. At the same time I drew attention to the fact that "clinical tests showed that the blood contains sufficient antitoxin (diphtheria) to afford protection." These were the days previous to the use of toxin-antitoxin*. Much to our surprise, some of these cases gave a negative Schick test in spite of the definite clinical signs of nasal diphtheria. In two instances the diphtheria bacilli were tested on guinea pigs and found to be virulent. Not long after these observations an infant died from diphtheria of the larynx which developed although the Schick test was negative. At postmortem a typical membrane was found on the larynx.

Since this time, there has been little opportunity to investigate this subject, as diphtheria has been banished from our institution by the routine use of toxin-antitoxin. Recently, however, we have met with three cases of nasal diphtheria which developed soon after their admission to the institution. These cases were characterized by the typical bloody nasal discharge. In two instances the cultures showed avirulent bacilli, in one which was obtained in December, 1930, the bacilli from the nose were virulent. In spite of this fact, not only was the Schick test negative, but tests carried out in February, 1931, with increasing doses of toxin 1/50-1/40-1/30-1/20-1/10-1/5 M.L.D. all failed to induce a skin reaction. As the result of these experiences we infer that a lack of the antiscorbutic vitamin exerts a local effect on the mucous membrane which diminishes its immunity, but at the same time may not be accompanied by a lowering of systemic immunity. It is probable that a similar phenomenon holds true in connection with a deficiency of vitamin A and that the marked changes in the epithelium, described by Wolbach, bring about a local diminution in resistance. Susceptibility to infections of the skin and of the respiratory tract which occur when this deficiency is marked may be largely a manifestation of a local pathological change.

*The runabout children had a habit of pulling off the buttons of their shoes and pushing them into their nostrils where they remained until they were removed some time later with forceps. This condition led to a bloody nasal discharge which on cultivation often showed diphtheria bacilli.

CONCLUSIONS

The antirachitic factor, whether given as ultraviolet irradiation, as irradiated ergosterol, or as cod liver oil does not increase the immunity of infants to respiratory infections.

Respiratory infections are not due to a lack of vitamin A and generally cannot be lessened by giving a diet rich in this factor even when supplemented with cod liver oil.

The average infant seems to receive an adequate amount of vitamin A in its milk, judging by the fact that xerosis of the eyes is exceedingly rare, and that no gain in weight or increase in immunity is brought about by adding vitamin to the diet. The same seems to hold true for older children and adults, in view of the infrequency of night-blindness, the first sign of this deficiency.

A lack of vitamin C may induce heightened susceptibility to infection of the respiratory tract. It may, however, induce merely local susceptibility without appreciable loss of systemic immunity. This peculiar phenomenon is manifested by the occurrence of typical nasal diphtheria, associated with virulent diphtheria bacilli, but a Schick reaction negative to highly potent solutions of toxin.

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Discussion

Dr. Hill: We have heard from Dr. Hess what I thought we would hear—a brilliant, intensely interesting paper.

The second part of the program departs from the traditional method of conducting a medical meeting. We have four pediatricians here, Dr. Utter, Dr. Garland, Dr. Percy and Dr. Romberg, whose experience, reputation and ability is such that what they have to say should carry with it considerable weight. Each one of these gentlemen will be asked four questions which he will endeavor to answer to the best of his ability from the background of his clinical experience.

The first question is as follows: From your clinical experience do you believe that cod liver oil does good to children over two years old who are free from active rickets and who are already taking a reasonable diet? If so, what good?

I will call first on Dr. Garland of Boston to answer that question.

Dr. Joseph Garland, Boston: I was hoping to be able to hear what my colleagues had to say before I was called on. Two or three years ago Dr. Holmes, who is the director of the laboratory of the E. L. Patch Company, published a paper on the use of cod liver oil among the employees of that company and demonstrated that those who took cod liver oil had fewer colds and also suffered less from dysmenorrhea. That was statistical. We do not have statistics and have to deal with our impressions. My experience is that I have never seen any value from cod liver oil in older children.

Dr. Karlton G. Percy, Boston: We are sacrificial lambs, and I think we have been goats. It comes down to the subject of what my opinion is. I believe that the medical profession is undoubtedly in the hands of the houses that are selling cod liver oil. We have it crowded down our throats or rammed underneath our skins so that we are led to believe that it is an absolute necessity. Practically speaking, when you have passed the infant age and a child is able to get a mixed diet, a normal child is in my opinion never in need of cod liver oil if he has a rational diet. I believe, however, that after infections, especially long drawn out infections, it does help a child to recover faster, and that it also helps perhaps during the winter months. My impression is that older children are perhaps less susceptible to infection, but that is only an impression.

Dr. Eli C. Romberg, Boston: It is difficult to tell when active rickets ceases. Empirically we say it ceases at about two or three years of age. Actually we are unable to determine when it does stop, however, unless we see the character of the epiphyses by x-ray.

Provided that the diet is adequate, I think that cod liver oil is unnecessary in the treatment of a child over two years of age. In certain cases, particularly after prostrating illness where the child has lost a lot of weight and is washed out, cod liver oil seems to do some good. I do not believe it helps because of its antirachitic value, but rather perhaps, because of the high caloric fat value contained in cod liver oil. The same results could probably be achieved by giving the child olive oil or some other fat product.

I believe that when children have teeth of poor quality, where decay is present and the color is poor, the administration of cod liver oil should be somewhat extended.

Dr. Henry E. Utter, Providence: I believe in the use of cod liver oil for children who are taking a reasonable diet. It increases the weight if the child is not gaining and certainly lessens the nervous irritability, which is an important factor in its use.

I do not believe that cod liver oil will prevent respiratory infections, but that the susceptibility to these infections is considerably lessened. Cod liver oil in addition to the regular diet is an important factor in preventing dental caries.

Dr. Hill: The second question is as follows: Do you believe in routine sunbaths, with the body completely exposed, for normal infants? If so, why? If not, why not?

DR. GARLAND: My answer to the question "If so, why?" is, I believe that the routine sunbath is of some value and advise my patients to adopt it; the reason why is because all the literature emphasizes its value.

DR. PERCY: If this refers to routine sunbaths, I should say "no" because sunbaths are baths offered by mothers at any time. I think the danger of sunbaths when there is no sun, which is the routine sunbath to the mother, is a definite danger; but there is no doubt that the child who basks in the summer sun, who has had a summer with very few clothes on, the infant especially, is in far better condition than the one who is kept under the sheets and who does not get the effect of the summer sun. However, as a routine procedure I should say "no".

DR. ROMBERG: I am assuming that you allow the youngster to go out in the sun whether he is dressed or not. Except that a child looks healthier by being tanned, and his appearance attracts more favorable comment from the neighbors, I do not see the necessity of exposing the child's complete body to the sunlight. If the mother has not given the child any cod liver oil, or cannot afford vitamin milk or viosterol, or is prejudiced against giving cod liver oil in the summer because she believes it overheats the blood, I think in such cases perhaps an exposure of the body to the sunlight is beneficial.

I think the risk of over-exposure of the child to the sun, bringing about blistered skin or gastro-intestinal upsets, is too great. In general, therefore, I do not see the necessity of especially exposing the body to the summer's sun.

DR. UTTER: I believe in giving sunbaths to infants. If we accept craniotabes as an early manifestation of rickets, and I believe we should, it is remarkable to note with what rapidity this symptom disappears. I make it a practice to stop cod liver oil when sunbaths are instituted. Under the use of sunbaths the children both eat and sleep better. The great danger of sunbaths is in overdoing the procedure. An exposure of ten minutes to the front and back daily is usually sufficient. If the skin becomes too pigmented we possibly produce a barrier to the entrance of the ultraviolet rays; if we can attain a general hyperemia, we are probably using a procedure of real value.

DR. HILL: The third question is as follows: Are you convinced that there is a detectable clinical syndrome prevalent in this country in young infants due to vitamin B deficiency? Have you seen any of the carbohydrates, with added vitamin B, do good to these infants or to any infants? If so, what good?

DR. GARLAND: I don't know how to answer that rather long question. I am open to conviction, but I am not convinced. I have not yet seen any sign of a detectable syndrome. If there is such a thing, I should be glad to know about it. I have seen no results from using preparations containing vitamin B, and I am not sure what they are.

DR. PERCY: Well, I think we have been sold by the pharmaceutical houses, first because of a very good piece of work by Dr. Dennett of New York, but thereafter taken up by wholesale houses. I am ashamed to say that I cannot see a syndrome which necessitates the use of vitamin B with cereal additions, though like the cases of difficult feeding in which the grandmother comes along and gives condensed milk and the baby does well, we do see infants do well with cereals. Whether it is due to the effect of vitamins or whether it is because of ignorance that it has been impossible for us to find the reason for it I don't know.

DR. ROMBERG: If we are to believe the investigations of the Toronto observers, there must be some such clinical syndrome as vitamin B deficiency. Up to this afternoon before I heard Dr. Blackfan's lecture about acrodynia which might possibly be traced to vitamin B deficiency, I interpreted the pallor and the loss of tone and the irritability and the crying as cramps and colic and teething, but now since I have heard this lecture I am going to be on the lookout for these vitamin B manifestations which so far we have not been able to recognize in the early form and which I personally have not seen. With the mixed diet that most of our patients are getting, it is hard to believe that a vitamin B deficiency could exist. I have tried the various dextri-vitavose preparations and I find that the children who do not take them get along as well as the children who do.

DR. UTTER: If vitamin B protects the nervous system there must be a great many children who show a deficiency of this vitamin, but so far I have been unable to recognize a distinct syndrome due to this deficiency.

I have but one observation to make in this respect. I use wheat flour in many formulae when a baby comes to me being fed on milk, water and sugar mixtures. There results a distinct lessening of nervous symptoms but it must not be forgotten that the mother or nurse is instructed in matters of routine, regularity of feeding hours, sunshine and sleep, all of which factors are important in making a baby happy. Under such circumstances it is hard to state just how much importance the addition of wheat has in the matter.

I have seen very little result in the feeding of preparations with addition of vitamin B not obtained by the use of other infant foods without this addition.

DR. HILL: The fourth question is as follows:

Do you think it rational to administer cod liver oil plus viosterol to normal infants? If so, why? If not, why not?

DR. GARLAND: I suppose sacrificial lambs should feel a sense of responsibility. Personally I can see no particular value in adding viosterol to potent cod liver oil. It may be a good thing, but I don't know.

DR. PERCY: At dinner we heard Dr. Hess speak about the unpopularity of cod liver oil, how little was taken because the baby didn't like the smell or because it was unpleasant for the mothers. Statistically he tried to show that about one-third of the cod liver oil ordered by milk stations was never given. And then he said that there was probably enough of the vitamins in milk if they were absorbed, to protect most children against rickets and vitamin deficiency. However, knowing the improvement that has occurred in the children of the United States in the last 25 years with the decrease of mortality and the increase in knowledge of the giving of cod liver oil, it does seem wise and just that every child should be ordered the proper amount of cod liver oil.

Now if human nature is such that what is ordered is not given in the proper amount, I have felt that if you can bolster up your cod liver oil with viosterol, you are going to get in a small dose which is going to be the necessary amount of vitamin a day, and I can say I have been using more viosterol with cod liver oil.

DR. ROMBERG: Ordinarily in normal infants the administration of a reliable plain cod liver oil is sufficient to take care of all the child's needs. However, when one realizes that so many of the cod liver oils on the market are poorly assayed or unstable or impotent, I believe that most of us feel more happy or assured if we can give the children cod liver oil supported by viosterol. Nowadays, however, it is hard to ask families to give their children viosterol and cod liver oil, with the prices that are being asked for such products.

DR. UTTER: I have never been quite convinced in the matter of using viosterol alone. To mix viosterol with cod liver oil does not seem a fair experiment for we never know which gives the desired result.

In 1929 I tried several experiments to satisfy myself in regard to the clinical value of viosterol and other antirachitics. Using craniotabes as the test, I gave one group of infants viosterol, another cod liver oil and still another cod liver oil with phosphorus. In the viosterol group it seemed to me that about as many developed craniotabes as I was accustomed to find in babies who had not received any antirachitic agent. In the cod liver oil group a smaller number developed craniotabes, while the group taking cod liver oil and phosphorus did not develop craniotabes at all. I have always believed that cod liver oil with phosphorus was the best antirachitic.

DR. HILL: We will now have a general discussion of the subject.

DR. RICHARD M. SMITH, Boston: After listening to the lambs I think we might go back to some of the ideas which Dr. Hess has been discussing. I am sure we have all of us been much interested in his presentation of the subject. There are two questions which constantly occur in the minds of us who are dealing with children and which are hard to answer. I can only ask them, as they are perplexing, though I have been considering them for years. These questions have been touched upon, but I think there are matters in doubt in relation to both of these questions.

The first has to do with the quantitative measurements of vitamins. We have been observing children for years before and since the addition of vitamins as such to the diet, though we all gave vitamins before we called them by that name. The question arises whether we have been treating our children with insufficient amounts of vitamins. If that is so, we ought to have seen and be seeing still a large number of children presenting evidences of vitamin deficiency.

It is true that scurvy is less common than it used to be, and we are all giving the children enough vitamin C to prevent scurvy. Yet with relation to the other vitamin I am not so sure, and I should like to have Dr. Hess say something about the quantitative measurements of vitamins from the clinical point of view.

The second question is closely related to that, and has been touched upon. We ought to know more about it namely, the possibility of our recognizing vitamin deficiencies in the early stages. It is easy to recognize a well-developed but not a latent case of scorbutus; and the same is true with relation to the other deficiency diseases. We recognize that night-blindness is an early symptom of vitamin A deficiency, but are there not other signs which are as recognizable as this and have a real significance? If we were properly informed and sufficiently alert, might we not detect some of these deficiency diseases before serious damage had been done?

I think that the discussion in relation to acrodynia is timely. It is true that we make the diagnosis more commonly than we used to do; probably this is because we now recognize the mild cases of the disease which were formerly undiagnosed. May it not be true also that there are children with vitamin deficiencies in small degrees or with slight manifestations, in which, if we knew more about the conditions, we might prevent more serious manifestations?

Dr. Edwin T. Wyman, Boston: From what Dr. Hess has told us, it seems to me that the way to keep our children's resistance to infection as high as possible is to keep them in the best possible condition by feeding them a proper diet and giving them sufficient Vitamin A, C and D.

Not so many years ago it was thought that if we fed fat, carbohydrates and protein in quantities suited to the individual child together with mineral salts contained in the food, that this was sufficient to supply all the needs for nutrition and development.

A few nights ago I heard Dr. E. V. McCollum speak and he said that there are 37 elements and chemical compounds all of which are necessary in the diet to promote proper growth and development.

After the second year if a child gets an ordinary American diet without too much sugar or wheat flour which would naturally replace other foods containing more of these necessary compounds, I believe one need think very little of vitamins or these various necessary chemicals. On the other hand, before a baby is two years old one has to think of vitamins, especially A, C and D. Before this last year I have thought that the possible lack of vitamin A in an infant's diet was of little consequence. However, Dr. Blackfan's cases at the Infant's Hospital this year have made me vitamin A conscious. One of the babies was fed on an adequate diet except that two ounces of cream were removed from the milk because of fat intolerance. This baby developed a respiratory infection and the autopsy showed a definite vitamin A deficiency. I think we should keep vitamin A in mind especially with babies who have a low fat tolerance and be sure that they get sufficient cod liver oil.

Vitamin C. I advise patients to get the best milk they can afford for their babies, certified milk if possible, and then either pasteurize or boil it for babies up to one year old. Of course this makes it necessary to consider vitamin C and see that they get sufficient orange juice or tomato juice to supply their vitamin C requirements.

In regard to vitamin D it seems to me that babies under two years of age should have vitamin D in addition to the vitamin D contained in their diet. There is no definite dose of cod liver oil, as babies' requirements vary according to the individual. As a general rule I think it is necessary to give two or three teaspoonfuls a day during the winter months and one to two teaspoonfuls during the summer months, depending in the summer on the amount of sunshine the baby gets. This amount of cod liver oil, however, isn't sufficient in all cases to prevent the occurrence of rickets and in some cases it is sometimes impossible to get the baby to take sufficient cod liver oil to prevent rickets. This is especially true in some very rapidly growing infants, babies born in the fall, twins and premature infants. In these babies the cod liver oil would have to be supplemented by viosterol, "Haliver" oil or sunlight. I believe the ideal food for a baby at the present time is vitamin D milk with sufficient cod liver oil added to take care of any possible vitamin A deficiency and orange juice to supply vitamin C.

I think that sunbaths are a good thing for all animals, if not overdone, and I believe the time will come when ultraviolet transmitting windows will be more generally used in nurseries, especially for babies born in the fall, premature babies and twins. Here the baby can have a sunbath in a warm room without the disadvantage of possible exposure to cold and I believe a half hour sunbath during the winter months gives the baby something he would not receive from cod liver oil or viosterol and would probably prove useful in maintaining his resistance against infections so common during the winter.

I would like to ask Dr. Hess if the cows fed on alfalfa give a milk more potent in vitamin A than do cows fed on ordinary hay.

Dr. John L. Morse, Boston: I feel thoroughly incompetent to discuss this question. There are undoubtedly such things as vitamins. I think what has been learned regarding the vitamins during the last decade has been one of the greatest advances in medicine for many years.

There is no question but that the vitamins are necessary for the well-being of infants and children. I suppose that this is true also in the case of adults. Personally I never worry about them. On the other hand, I am thoroughly convinced that an insufficiency of any or all of the vitamins in early life is much less common than is generally believed. I think that the frequency of an insufficiency of the vitamins has been exaggerated, probably largely because of the advertising of the commercial houses. In that connection we must be extremely skeptical about believing anything that we read either in the newspapers or in the medical journals and we must wait for work like that of Dr. Hess before we accept anything. Work like his we can believe, for it is founded on facts and observations. Most of the stuff that we read is simply hot air.

Another thing I think we should be very skeptical about is the application of the results of animal experiments to human beings. Do not misunderstand me when I say this. I thoroughly believe in animal experimentation, and we would not know what we do about vitamins now if it had not been for animal experimentation; and a great deal of what we will know in the future will be learned from animal experimentation. Nevertheless, we must be careful as to how we apply those results to infants as Dr. Hess has

said. As I see it, what is most needed at present is an exact knowledge of how much of each vitamin is needed, and what the early symptoms of a slight deficiency of each of the vitamins are.

Dr. Alfred Hess (closing): The first question is as to the quantitative requirement of vitamins clinically. In regard to vitamin A I should think that most children, really almost all children, are getting enough. About the only way you can feed an infant is by giving milk which contains about 2,000 units to a quart. That ought to be enough.

There may be conditions where vitamins are not utilized; for instance, we do not know the relation of fever to their utilization. It may be that if a child has fever, it consumes a great deal of vitamin. It is quite possible that if the child vomits or has bowel disturbance it may not get the requisite amount of vitamins and one might have to increase the quota during an attack of this kind.

Again if the milk isn't pasteurized sufficiently, the vitamin A may be lost because of its susceptibility to oxidation; prolonged oxidation may destroy the vitamin. There is a method of homogenizing milk by beating it up and aerating it. That is a dangerous procedure from the standpoint of vitamins, both A and C. In Switzerland they had almost no scurvy until they introduced this method of mixing the milk.

In regard to the vitamin B, I think there is a sufficiency. I know of no symptoms due to a lack of vitamin B. I have tested various preparations and found them of no advantage. I tested vitavose and various extracts of wheat germ and potent extracts of yeast.

We see very little scurvy. I understand it is increasing somewhat, but I always believe that if we have a considerable number of any one of these deficiency diseases manifest, as well as latent cases, it must not be infrequent. It is hard to believe that there are latent cases of scurvy by the hundreds. The same is true of vitamin A. Xerosis of the eyes is rare and it is hard to believe that there are hundreds of latent cases. Wherever latent beriberi occurs, as in Japan, there is always a parallelism between manifest and latent cases.

In regard to vitamin D I do think we need a supplement, for milk has practically no vitamin D. The more milk you give, the less the scurvy, but the more you overfeed a child with milk, the more you get rickets; so there must be very little vitamin D in milk. Cod liver oil in three teaspoonful doses daily protects infants generally.

I do not quite agree with Dr. Utter in regard to craniotabes. This lesion is peculiar. Rickets is a lack of calcification of the cartilage; that is its main manifestation. The epiphyses do not ossify. Now the cranial bones do not contain cartilage, and we should not take a membranous bone as a criterion in considering rickets.

Premature babies must be regarded as entirely different animals. You may have an agent that will work beautifully on the normal infant but will not work on the premature.

I was asked about the milk of cows fed alfalfa. I don't know whether cows which get alfalfa have more vitamin A in their milk. Rapidly dried clover hay has a great deal more vitamin A than that slowly dried. You can't increase it beyond a certain amount. We gave yeast to cows, the yeast being rich in vitamin B, but in giving that amount of yeast which has vitamin G also, I found that neither had been increased, so that whether feeding alfalfa to the cows causes an increase in vitamin A remains to be proved.

In regard to animal experimentation, we have to be very careful. For example, one needs forty to fifty times more irradiated ergosterol for chickens than for rats in order to get the same result.

Another matter should be mentioned; you can't talk about units of cod liver oil and irradiated ergosterol and irradiated milk because they are different. You cannot compare irradiated ergosterol with irradiated milk. I have found that milk from cows fed with irradiated yeast has few "rat units". You have to give 800 units of irradiated ergosterol to protect an infant, and 160 units of cod liver oil and 160 units of this vitamin D milk, so that you can't speak in terms of units except for a particular preparation. This shows that they are utilized differently or bound differently chemically.

About the recognition of the early stages of deficiency-disorders; that is what we need in order to recognize them. The chemical test for rickets is lowered phosphorus in the blood. It is possible that we may find some chemical tests to recognize early deficiencies in relation to a lack of the other vitamins.

ACUTE RECURRENT INTUSSUSCEPTION IN CHILDREN*

Case Reports

BY AUGUSTUS THORNDIKE, JR., M.D.†

THE operative treatment of intussusception has been known and practised ever since 1873, when Lichtenstein[1] reported his first laparotomy. However, owing to the high mortality, it was put in disrepute soon afterwards. In 1885, Braun[2] introduced this method of treatment again and ever since that time it has remained the most popular form of treatment. Hirschsprung[3] in 1905 reported his successful treatment of intussusception "auf Wasser reponierte" and ever since then there has been a controversy as to which method gives the best results. Monrad[4], Hipsley[5, 8], Arntzen and Helsted[6], Olsson and Pallin[7], Brandberg[9], and a few others have advocated the use of non-operative treatment, but after a brief and unsuccessful trial, the surgeons of the Children's Hospital have discarded it. The treatment used most successfully ever since the first case (in 1889) of intussusception at this clinic has been operative. The stimulus that formed the basis of adoption of this form of treatment was Clubbe's[59] monograph.

TABLE I

INCIDENCE OF INTUSSUSCEPTION AT THE CHILDREN'S HOSPITAL, BOSTON

1869-1888	0	1914	11
1889	1	1915	16
1890-1897	0	1916	12
1898	2	1917	14
1899-1900	0	1918	14
1901	1	1919	20
1902	1	1920	12
1903	1	1921	26
1904	3	1922	28
1905	1	1923	19
1906	2	1924	28
1907	2	1925	25
1908	6	1926	24
1909	4	1927	18
1910	7	1928	22
1911	7	1929	19
1912	7	1930	15
1913	13	1931	23
1932 (to date)	1		

Total Number of Cases	405
Recurrences	5
	400

Since this hospital opened its doors in 1869, there has been a total of 405 cases admitted. It is interesting to note that of this total five have been postoperative recurrences. Subtracting the operative recurrences from the total, we obtain a total number of primary intussusceptions of 400. This gives a ratio of one recurrence to every 80 cases. From the literature reports as shown in Table II below, this ratio appears conservative. However, statistics are always confusing and here it is obvious that the series reported are in many instances too small. Hess'[12] ratio of 1:114, and Cohen's[13] ratio of 1:10 represent the two extremes of incidence in acute recurrent intussusception.

Cohen[13] made a careful review of the literature in his article in 1921, and since then later reports have brought in a total of 75 cases of operative or proved recurrent intussusception reported in the literature. This figure excludes all adult intussusception recurrences and does not include cases reported twice as two. It has been difficult to isolate cases from the literature that could be counted in this total, and for that reason there may have been a few cases overlooked. No case of unproved intussusception has been included and no case of chronic recurring intussusception. This is assumed to be a fairly accurate figure.

The etiology of acute recurrent intussusception is oftentimes as obscure as in simple acute intussusception. Sometimes, however, definite pathology can be demonstrated. It must be remembered that any factor that causes acute intussusception may likewise produce a recurrent intussusception. Therefore, for simplification the classification in Table III is presented.

Volumes have been written on the etiology of intussusception, but inasmuch as this paper is a report on acute recurrent intussusception, the explanation of the factors present or absent in the causation will not be discussed. Any reader may consult the literature references. To the 75 cases already reported (Table II) five more are here added from the records of the Children's Hospital:

(1) F. A., No. A-105437, a female child of six months was admitted to the hospital May 23, 1927. Family history and past history were negative. The chief complaint was blood in the stools following cramps two hours previous in a well baby. About 7 A. M. on the day of admission, the child had cried with pain and drawn up its legs. About 7:30 A. M. mother gave the child an enema which was returned immediately afterward, blood-tinged. The child vomited two hours later and the physician referred the patient to the hospital. There had been no more blood after 7:30 A. M.

Physical Examination: A well-developed and nourished child was admitted looking fairly ill. The examination was negative except for the abdomen. This was normal in contour and presented very slight muscular spasm on deep palpation. No definite mass was palpable. Rectal: No tumor was

*From the records of the Children's Hospital, Boston. Demonstration of 4 cases at the meeting of the New England Pediatric Association, May 6, 1932.

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palpable but slightly blood-tinged mucus followed withdrawal of the finger; this resembled "prune juice".

Admitting Diagnosis: Intussusception.

Operation: Reduction for Intussusception.

May 23, 1927. Through a 2½ inch right rectus muscle splitting incision, an ileocecal intussusception was found having advanced into the transverse colon about two inches beyond the hepatic flexure. Without difficulty this was reduced. The wound was closed in layers.

Progress Notes:

The patient made an uneventful convalescence and was discharged home on the ninth day with the wound well healed.

Discharge Diagnosis: Intussusception.

READMISSION:

This patient was readmitted on August 8, 1927, with a history of having been perfectly well following operation until the previous night, when she had cramps. The child refused food but drank a little water. One-half hour before readmission the child had passed a bloody stool.

Local Examination: The abdomen was soft without spasm or tenderness. No masses were palpable. Rectal: The examining finger was covered with blood upon withdrawing it from the rectum. Temperature 101°.

Admitting Diagnosis: Recurrent Intussusception.

Operation: Reduction.

August 8, 1927. A right rectus muscle splitting incision was made at the level of the umbilicus. On opening the peritoneum a small amount of slightly blood-tinged fluid escaped. The mass was palpated at the mid portion of the transverse colon and was reduced intra-abdominally as far as the cecum. The cecum was then delivered into the wound and reduced completely to the ileocecal valve. The serosa had not been torn. The intestines quickly returned to normal color. The incision was closed in layers without drainage.

Progress Notes:

The child ran an unexplained fever for five days after operation and otherwise made a perfectly uneventful convalescence. Discharged on the twelfth day with the wound clean and healed.

Discharge Diagnosis: Recurrent Intussusception.

Note: This case is of interest because intussusception occurred twice within three months.

(2) B. T., No. A-75492, a female child of eleven months was admitted to the hospital October 4, 1924. Family history and past history were entirely negative. At about 5 P. M. on the day of admission, the child suddenly became ill and acted as though in pain. She vomited soon after, had a bloody stool at 6 P. M. and another at about 7:10 P. M. The local physician

TABLE II

TABULATION OF REPORTED ACUTE RECURRENT INTUSSUSCEPTION IN CHILDREN

Author	Number of Intussusceptions	Number of Recurrences	Etiological Factor
Hirschsprung[3]	107	3	
Monrad[4]	107	6	
Arntzen & Helsted[6]	2	1	
Hipsley[8]	51	2	
Hess[12]	1028	9	
Cohen[13]	41	4	
Richardson[14]	75	2	2 Tumors (Adenoma)
Clubbe[15]	144	2	
Adams & Cassidy[16]	100	1	
Mahon[17]	3	3	
Turner[18]	1	1	Tbc. Enteritis
Eccles & Laidlaw[19]	89	1	
Allen[20]	27	1	
Peskind[21]	1	1	
Graham[22]	1	1	Tumor (Small Round Cell Sarcoma)
Hawe[23]	1	1	
Clarke[24]	1	1	
Barrington-Ward[25]	1	1	Tumor (Adeno-papilloma)
Bennett[26]	1	1	
Thorner[27]	1	1	
Willis[28]	2	2	Tumor—1 Adenoma —1 Fibroma
Romanis[29]	374	5	2 Meckel's Diverticula 3 Adenoma
Porter[30]	11	1	
Gould[31]	1	1	
Dowd[32]	1	1	
Cleveland[33]	1	1	
Bolling[61]	50	1	
Abadie[62]	1	1	
Herbert[63]	1	1	
Nixon[64]	1	1	
Mühsam[65]	1	1	Meckel's Diverticulum
Kellock[66]	1	1	
Reisinger[67]	42	1	Polyposis Intestine
Close[10]	363	14	
		75	

was called and referred the patient to the hospital.

Physical Examination: Negative except for the abdomen, which showed some dullness on the right side with definite resistance and an indefinite mass in this region.

Admitting Diagnosis: Intussusception.

Operation: Reduction.

October 4, 1924. A right rectus muscle splitting incision was made. The peritoneum was opened with the escape of a small amount of clear fluid. The intussusception was reduced intra-abdominally except for the last three or four inches. The cecum was then delivered into the wound and the reduction completed. The wound was closed in layers without drainage.

Progress Notes:

Except for a high temperature for the first 48 hours, the child made an uneventful recovery.

Discharge Diagnosis: Intussusception.

READMISSION:

This patient was readmitted on December 7, 1924, with a history of having been perfectly well since the operation for intussusception in October. On the morning of readmission the child was fussy and vomited once. About 3 o'clock the patient appeared to have cramp-like pains in the abdomen and the mother noticed a slightly blood-tinged stool. The child vomited again. The baby was seen about 5 o'clock in the afternoon; did not look very ill; temperature 99°.

Local examination: During the examination the baby had some cramp-like attacks of pain, vomited once, and passed considerable blood by rectum. Operation advised.

Admitting Diagnosis: Recurrent Intussusception.

Operation: Reduction.

December 7, 1924. The incision was made through the old scar. A typical ileocecal intussusception which had advanced almost to the hepatic flexure was easily reduced. No mechanical cause was found for the recurrence. The wound was closed in layers, the old scar being excised first.

Progress Notes:

A Levin tube was placed in the stomach on the operating table and left in for twelve hours. The child made an absolutely uninterrupted and normal convalescence and was discharged on the twelfth day.

Discharge Diagnosis: Recurrent Intussusception.

Note: Recurrence within two months.

(3) G. N., No. 144413, a female child of eight months was admitted to the hospital December 2, 1930. The family history and past history were negative. She had been taken from the breast one week previous to admission and put on a formula which did not agree with her. The bowels were constipated and hard movements were passed after considerable straining. Some blood was noticed with a movement about four days before admission. The child took feeding quite well this morning but about 2 P. M. appeared to have some abdominal discomfort, vomited a little and passed a bloody stool. The vomiting recurred in about one-half hour and another bloody stool was passed. The mother stated that she noticed a protrusion from the anus "like bowel com-

TABLE III

CLASSIFICATION OF ETIOLOGICAL FACTORS CAUSING INTUSSUSCEPTION

Cause Explainable by No Pathological Findings	*Cause Explainable by Definite Pathological Findings*
I. Nothnagel[34] theory of spasm and irregular peristaltic movement in intestinal wall, produced by faradic current in dogs.	1. Extra-intestinal Pathology. 1. Enteric Cyst (Bryan[38]) 2. Omphalo-mesenteric Cyst (Coutts[39]) 3. Adhesions (Alexander[40])
II. Fitzwilliams[35] explanation of difference in rapidity of growth in cecum and ileum of young babies.	II. Intra-Intestinal Pathology. 1. Foreign Body in Intestinal Lumen. a. Toy (Teddy Bear)—(Haydon[41]) b. Round Worms (Keane[42])
III. Alvarez[36]—Antiperistalsis does occur normally.	2. Congenital Abnormality in Intestinal Wall. a. Meckel's Diverticulum (Romanis[43], Lower[44], Hess[12]) b. Congenital Stenosis of Ileum (Warwick[45])
IV. Shaw[37]—Peristalsis vs. antistalsis normal physiological muscular activity in ileocecal region.	3. Infections. a. Tubercular Enteritis (Turner[46]) b. Infectious Diarrhea (Fitzwilliams[47])
True cause probably a combination of 1, 2, 3, and 4.	4. Tumors in the Intestinal Wall. a. Mucous Polyp (Bevers[48], Reisinger[67]) b. Lipoma (Summers[49], Heinonen[50]) c. Fibroma (Rossoni[51], Clifton and Landry[52]) d. Fibro-Adenoma (Oden[53]) e. Lymphosarcomatosis (Green[54]) f. Adenoma (Willis[28]) g. Leiomyoma (Green[54]) h. Sarcoma (Graham[22]) i. Mucous Cyst of Cecum (Bazin[55], McAuley[56])

In children pathological findings are relatively rare, but they have been reported as causing recurrent Intussusception as noted in Table II.

ing down," but this went back and was not seen again. The vomiting continued at intervals, and before admission the child had had about 8 stools of blood and mucus.

Physical Examination showed a well-developed and healthy looking female infant. She did not appear to be in great pain but resented examination and appeared to have slight abdominal pain at frequent intervals. The child immediately became rigid when examination of the abdomen was attempted; there was no undue fullness present and no dullness or tympany. When under the anesthetic a mass could be felt in the left flank and left iliac fossa, apparently the colon. Rectal: A soft, rounded mass could be felt about 2 inches from the anus; this was movable and could be circumscribed while the finger could be passed into a dimple on the surface. Blood and mucus were discharged from the rectum after the examination.

Admitting Diagnosis: Intussusception.

Operation: Reduction.

December 2, 1930. There was a very definite sausage-shaped mass felt to the left of and below the umbilicus. Through a four inch mid-right rectus muscle-splitting incision, a large intussusception was seen and felt in the pelvis and left lower quadrant. This was reduced intra-abdominally until the advancing point was beneath the incision; then the mass was delivered and complete reduction easily accomplished. The bowel was in good condition. This was not the usual ileo-colic type but colon into colon. The starting point was in the wall of the ascending colon. There was an unusually long colon with very loose mesentery. The usual enlarged mesenteric glands were seen and felt and there was no excess free fluid. The wound was closed in layers.

Progress Notes:

The patient made an uneventful convalescence and was discharged home on the tenth day. The wound was well healed.

Discharge Diagnosis: Intussusception.

READMISSION:

The patient was readmitted on January 27, 1931, having been fairly well since discharge from the hospital six weeks ago. This morning the child took her 5 o'clock feeding quite well but at 8 o'clock began to cry as if in pain and about five minutes later started to vomit. She vomited three or four times. Before 8 o'clock the child had a normal bowel movement, but about 20 or 25 minutes after crying started she had a stool which showed mucus and blood. The parents were advised by the physician to bring the baby to the hospital immediately.

Local Examination: Child tended to hold the right side of her abdomen rigid. A mass could be felt in the right upper quadrant. Rectal: No mass felt; blood was present on withdrawing the examining finger.

Admitting Diagnosis: Recurrent Intussusception.

Operation: Laparotomy and Reduction of Intussusception.

January 27, 1931. The scar in the skin of the previous operation was excised in a narrow strip. The anterior layer of rectus sheath was opened and the rectus muscle retracted outward. There was a moderate amount of free, clear, straw-colored fluid. The advancing point was found low in the pelvis. Reduction was easily accomplished by pushing the advancing point back, intra-abdominally, until the cecum was reached when the mass was delivered into the wound and complete reduction made under direct vision. There was considerable edema of the ileo-cecal valve but no tumor could be made out. The usual enlarged mesenteric glands were present. No cause for recurrence was determined. The wound was closed in layers.

Progress Notes:

The patient made an uneventful convalescence and was discharged on the ninth day.

Discharge Diagnosis: Recurrent Intussusception.

Note: Recurrence within two months.

(4) M. P., No. 132102, a female child of seven months was admitted to the hospital September 29, 1929. Family history and past history were negative. About eleven hours before admission, the child suddenly became markedly irritable and cried out in pain (1 P. M.), flexing her thighs on the abdomen. An enema was given without relief. Vomiting started about 4 P. M. and the patient vomited eight times. About 4 P. M. a physician was called. Shortly after, the child had a bloody stool. The pain and vomiting persisted and admission to the Children's Hospital was advised.

Physical Examination: A well-developed and nourished child in no apparent distress from abdominal pain. Abdomen showed no marked spasm. A mass about the size of a large sausage could be felt in the right upper quadrant and extending across to the left. Rectal: No mass palpable but blood was present on the examining finger.

Admitting Diagnosis: Intussusception (Ileocolic).

Operation: Reduction.

September 30, 1929. A high paramedian right rectus incision was made through the skin and carried down to the muscle; the muscle was split. A large tumor mass could be felt. It started at the hepatic flexure and extended to the splenic flexure. The leading point at the splenic flexure was delivered into the wound and by careful manipulation was forced to the right and downward. The ileum was carefully and fully expressed at the ileocecal valve. The cecum was normal in appearance except for marked edema and redness. The abdominal cavity was filled with normal saline. The wound was closed in layers.

Progress Notes:

The child made an uneventful recovery and was discharged home on the tenth day.

Discharge Diagnosis: Intussusception (Ileocolic).

READMISSION:

The patient was readmitted on May 12, 1931. She had been perfectly well since discharge eight months before until 17 hours before admission, when she woke up with pain. The mother thought it was a cramp and gave her a glycerine suppository which brought forth a natural and normal movement without pain. During the rest of the night the baby was very fussy and vomited ten or twelve times. The family physician was called and advised a suppository in the middle of the afternoon. This resulted in a bloody stool. As soon as the mother saw this, she recognized the possibility of intussusception and brought the child directly to the hospital.

Local Examination: A well-developed and slightly undernourished child of two years was lying on the examining table with knees drawn up. The abdomen was soft, but not easily palpated, with an old operative scar in the right lower quadrant. A mass the size of a walnut could be felt just below and to the left of the umbilicus. It was soft, not tender, and quite movable. The entire transverse and descending colon seemed full. Rectal: Large amount of blood passed by rectum. Anal sphincter tight. There was a ring-like constriction at the ano-rectal junction. Polypoid mass felt by tip of finger.

Admitting Diagnosis: Recurrent Intussusception.

Operation: Laparotomy and Reduction of Intussusception.

May 12, 1931. The lower portion of the old scar was excised and incision extended downward, making a four inch low right rectus muscle splitting incision. Peritoneum was opened carefully with the escape of a small amount of free fluid. There was some adherent omentum to the scar of the previous opera-

tion. An extensive intussusception was found with the leading point down in the pelvis, practically to the rectum. Reduction was accomplished intra-abdominally without any difficulty until the region of the ascending colon was reached. Here the mass was delivered and found to be extremely edematous. It was bathed with hot saline and complete reduction finally accomplished without injury to the bowel wall. The cecum was so edematous it was almost white in color, but the bowel was considered viable, so it was replaced within the abdomen. The usual associated enlarged mesenteric glands were found. The intussusception was of the ileocolic variety and no definite cause for its recurrence could be made out. The wound was closed in layers.

Progress Notes:
Discharged on the tenth day after an uneventful convalescence.

Discharge Diagnosis: Recurrent Intussusception.
Note: Recurrence within eight months.

(5) R. R., No. 137567, a male child of thirteen months was admitted to the hospital on April 14, 1930. Family history and past history were negative. About seven hours before admission (4 A. M.) the child woke the family by screaming and cried hard for one-half hour. An enema was given and a normal movement resulted. One-half hour later there was another similar episode. There was no vomiting. While in the examining room, he passed a green movement streaked with blood.

Physical Examination: The patient was a well-developed and nourished infant. Examination suggested a mass in the right upper quadrant. Rectal: Blood-streaked stool specimen presented after withdrawal of examining finger.

Admitting Diagnosis: Intussusception.
Operation: Reduction.
April 12, 1930. A right rectus muscle splitting incision was made. A small amount of straw-colored fluid escaped. An intussusception had advanced as far as the hepatic flexure. This was easily reduced. The bowel wall was somewhat edematous but perfectly viable. The wound was closed in layers.

Progress Notes:
Except for a temperature of 104° for the first 48 hours, the child made an uneventful convalescence.

Discharge Diagnosis: Intussusception.

READMISSION:

Readmitted on February 26, 1931. The child had been perfectly well since discharge until 18 hours before admission when suddenly, after eating supper, he doubled up with acute abdominal pain. He had several paroxysmal attacks of acute abdominal pain lasting from two to five minutes. He vomited his supper. He was given an enema one-half hour after onset with good result; no blood. He was given milk of magnesia (twelve hours before admission) and mineral oil (six hours before admission) and had had four scant, loose movements, brownish and mucoid; the last two contained a small amount of blood.

Local Examination: The child held his legs flexed over his abdomen and appeared to be in acute distress. The abdomen was slightly distended with gas and tympanitic throughout. The child cried and struggled and the abdominal examination was not satisfactory. There appeared to be slight tenderness in the right lower quadrant on deep palpation. The old operative incision slightly to the right of the midline was well healed. No masses, fecal material or tenderness present. Rectal: A small amount of brown fecal material and bright red blood was present on the examining finger.

Admitting Diagnosis: Recurrent Intussusception.
Operation: Exploratory Laparotomy.
February 26, 1931. A right rectus muscle-splitting incision was made just outside the old scar. A small amount of free, clear fluid escaped. The ascending colon was very edematous and evidently there had been a definite intussusception, which was partially reduced and the reduction completed when the cecum was delivered into the wound. No mechanical cause was found for the recurrence. The wound was closed in layers.

Progress Notes:
Discharged home relieved on the ninth day after an uneventful convalescence.

Discharge Diagnosis: Recurrent Intussusception.
Note: Recurrence within ten months.

The symptomatology in each of these five cases was precisely the same in the recurrences as in the initial attack. As a result of this situation, the child's condition was recognized and the patient was brought to the hospital early. Naturally, under these circumstances, the mortality was low, and in all five instances there was one hundred per cent recovery. None of our cases recurred more than once; none had gangrene or tumor requiring resection; but Barrington-Ward[25] reports one case of a seven year old child that had seven recurrences in a period of two years after the first laparotomy, where definite pathology was found after resection (adeno-papilloma). The time interval between recurrences seems to vary a great deal. The longest interval between recurrence and the original attack was reported by Arntzen and Helsted[6], eight years; and the shortest by Cohen[13], thirty hours after the first operative reduction. On the average, all of our cases recurred after a five month interval, the longest interval being ten months and the shortest, two months. None have recurred more than once, and in all of our cases no explanation was discovered of pathological nature.

The diagnosis of acute recurrence should be made easily, but it is well to recall, as Gallie[58] has pointed out, that other conditions may produce the same symptomatology. The presence of a tumor and "prune juice-like" bleeding on the rectal examining finger is pathognomonic. Arntzen and Helsted[6] have indicated that x-ray shows a definite picture in all cases. Some of the conditions which may be confused with intussusception are the following: dysentery, ulcerative colitis, Henoch's purpura, Meckel's diverticulitis, hemorrhagic pancreatitis, and intestinal obstruction due to mechanical or congenital abnormalities within the peritoneal cavity.

The treatment of recurrent intussusception is operative reduction. However, if other pathological conditions are found causing the intussusception, they may be dealt with at the same time provided the condition of the patient warrants it. A resection on a small infant is attended by a very high mortality, and unless definite gangrene of the bowel is present, it is always better not to remove any intestine after the intussusception has been reduced. Even a Meckel's diverticulum, should it be demonstrated

as the etiological factor in causing the recurrence, is better left in situ and removed at a later date. Sometimes, however, the diverticulum or a portion of it remains intussuscepted or is gangrenous, and in that case a diverticulostomy through a stab wound is the safest immediate procedure. If resection must be resorted to, an enterostomy is the procedure of choice.

The technical difficulties of intestinal suture into edematous bowel on a small infant are so ominous and recurrence is so rare in the experience of this hospital that the surgeons do not feel warranted in changing their operative procedures of reduction to include an anchor suture between the cecum and ileum as suggested by Lamson[11].

The author wishes to take this opportunity to thank Dr. G. D. Cutler and Dr. T. H. Lanman for the privilege of including their cases in this report.

CONCLUSIONS

1. Reports of five cases of acute recurrent intussusception are recorded. A ratio of acute postoperative recurrence of 1:80 acute intussusceptions is found in a total of 400 cases from the Children's Hospital.
2. In this series of acute recurrent intussusception cases, the longest interval between the first and second attack was ten months; the shortest was two months, and the average was five months. No case recurred more than once.
3. A total of 75 cases of acute recurrent intussusception in children is collected from the literature.
4. The symptomatology and differential diagnosis are discussed.
5. The treatment of recurrent intussusception should be surgical. The operative results in this series of five cases were one hundred per cent recovery.

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THE INFANTILE ECZEMA PROBLEM*

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IT is probably true that in infantile eczema we are dealing with a condition which is produced not by one, but by many factors, although the majority of cases are caused by much the same mechanism. Any irritant, whether exogenous or endogenous, may cause eczema, and future efforts in investigation must be directed to an attempt to determine as many of these irritants as possible, and to detach from the large group with unknown etiology as many smaller groups as possible with known etiology. This is what has been going on in the study of adult eczema for many years. Psoriasis, scabies, pityriasis rosea, and epidermophytosis were all once called "eczema". There is a feeling among dermatologists at present, largely due to the influence of Bloch of Zurich, one of the world's keenest students of eczema, that most cases in adults are due to external irritants, specific for the individual. He places eczema in the same class as dermatitis venenata, a contact dermatitis, and finds that the irritants which may produce it are innumerable.

It is very difficult, however, with infants, to prove that the eczema has its inception from external agents. True, every case is aggravated by external insult, but it is unusual to find a severe, typical case where any external agent can be proved the prime offender. One would expect this to be true, for the baby is exposed to comparatively few external irritants. An exception may be mild eczema of the cheeks, caused by water, soap, winds, and changes in temperature.

The rôle that fungi may play has been pointed out by Schwarz and White of this city, and by Cleveland White of Chicago. They have isolated these organisms from many cases of infantile eczema, but believe that their rôle in pathogenesis is not yet quite clear. It is somewhat difficult to determine clinically what may be a fungus case and what not, for White and Schwarz say that this is never a moist or vesicular disease, and Cleveland White says that fungi are most likely to be found in moist, oozing eczema of the face. They are agreed that fungi are especially likely to be associated with the type of eczema that persists into childhood and that the lesions may become much thickened and infiltrated, such as one sees so often in front of the elbows and in back of the knees in children from three to five years of age. We have treated a number of cases on this basis, with Schwarz's ointment of mercurochrome and salicylic acid, and have obtained better results with it than with any other. Crude coal tar is likewise a fungicide, and it may not be impossible that part of the good results often obtained with tar are due to its power in this respect. There can be no question that fungi play a part in a certain number of cases of infantile "eczema". How important this part is, is not easy to determine, for fungus infection may simulate in appearance almost any form of eczema. Undoubtedly, a large amount of what we see in infantile eczema is entirely secondary to the original process, and results from scratching, rubbing, oozing and crusting. Such a soil is ready for any sort of infection, and it is probable that low-grade infection often plays an important rôle in keeping the process active.

Most of the evidence we have points, however, to some internal agent or process as a primary causative factor in the majority of cases, and it is my purpose to discuss some of the possibilities.

One of the first clues was furnished by Talbot and Towle about twenty years ago when they found that some eczematous infants had excessive amounts of fat in the stools, and that their skin condition improved when the fat in the diet was reduced or omitted. They were very modest in their statements, and said definitely that fatty indigestion dealt with "but a small phase of the general problem of infantile eczema." Yet for many years afterwards, fat was removed from the diet almost as a routine measure, and still is today to a certain extent, often with unsatisfactory results to the nutrition of the baby. It seems fair to say that such procedure is desirable, and is likely to be beneficial provided the baby is being overfed with fat; but at the present time, when most babies are fed on whole milk dilutions, it is not so very common to find one who is fed on too large amounts of fat, or where the fat seems to be a factor in the production of the eczema. It is well to investigate the fat intake and fat digestion, but wise to proceed cautiously in removing it from the diet, unless there is gross overfeeding.

In view of our modern knowledge concerning

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the rôle of unsplit protein in causing eczema, it may well be that excessive amounts of fat in the diet do not operate primarily, but in a secondary manner by preventing efficient stomach digestion of protein.

The next ray of light shed upon the subject was by Schloss, about 1914, when he demonstrated that eczema and asthma might be caused by specific sensitization to foods or other substances, and that the offending agent might be detected by means of the skin tests. Blackfan, in 1916, was the first to apply this procedure to an extensive series of infants with eczema, and found positive skin tests in about sixty per cent. Since then skin testing has become an accepted part of the program in trying to get at the disease, and has been widely employed in this country, but has never found great favor in Europe. There is no question whatever that a series of eczematous infants will give a higher percentage of positive skin tests than a series of normal infants, and since 1916 many such series have been reported, with positive tests ranging from forty per cent to eighty-five per cent. In infants with eczema, the usual reaction is an area of erythema about the scratch, without a wheal. Large wheals may occur, however, and usually indicate a more severe sensitization. The interpretation of skin tests in eczematous infants is not entirely satisfactory, because their skins are very sensitive, and react easily to every stimulus. Sometimes large areas of erythema are obtained to every test applied including the control. Bloch has shown that the skin of eczematous persons is many times more sensitive than that of normal individuals. It is of some importance to make scratches of as nearly the same size and depth as possible, so that error incident to variations in trauma will not be introduced, and for this purpose we have devised a scarifier constructed from a small leather punch, which makes a linear circular cut, naturally always of the same length. With this technique, if there is around one or more scratches an area of erythema much larger than around the others, we call these positive. If there are large areas of erythema around every scratch, of about the same size, we pay no attention to any of them. What one often gets are areas of erythema of varying sizes around nearly all the scratches, and interpretation is difficult and unsatisfactory. Skin testing, in eczematous infants, is not an accurate procedure, and one must not take it too seriously.

What is produced by means of the skin test is a temporary dilatation of the small cutaneous vessels, giving erythema, and if a wheal occurs, it simply means that the substance with which the test was made has acted so strongly that it allows plasma to exude through the vessels and produce a wheal. The reactivity of the small arterioles and capillaries of the skin to various stimuli has a great deal to do with infantile eczema, and the solution of the problem may lie in a more complete understanding of this. What we really do in making a positive skin test is to reproduce on a small scale probably much the same mechanism that is involved in the production of the eczema, and it is probable that the first step in the eczema is a capillary dilatation, which goes on later to exudation, crusting, and gradual infiltration of the surrounding tissue.

We see undoubtedly a certain number of cases which give very definite skin reactions to one or more foods that are being eaten, and that the skin condition is due to ingestion of this food or foods is proved by its prompt clearing up when these are omitted from the diet. We see frequently, however, either negative tests to food proteins, or a series of rather indefinite tests to many proteins, some of which may have been eaten, some not, and we often see no improvement in the eczema when these are removed from the diet. We can explain only a comparatively small number of cases of infantile eczema by a specific sensitization to one or more definite foods, and may it not be quite possible that we are sometimes dealing with an irritation caused by sensitization to some substance or substances which may be produced from *any* food protein, or that poor or incomplete digestion of protein in general, with resultant absorption of intermediate protein split products, may be a cause? Histamine, which is such a powerful vasodilator that it gives a markedly positive skin test when applied to any normal skin, is formed from the amino acid histidin, and histidin is a constituent of practically all food proteins. Furthermore, there are many other protein decomposition products formed within the digestive tract, which might well cause skin damage when absorbed. There is considerable food for thought in this, and we are trying at present to isolate from the stools of eczematous infants, an extract which will give positive skin tests, so far with very meager results.

In the very worst cases there is an intense diffuse redness, not in patches, but covering almost the entire body surface*. In some parts there may be crusting and oozing, in others the skin may be relatively smooth. The hands and feet are *very moist, very cold, and blue,* indicating marked vasomotor dysfunction. The redness disappears very readily wherever a drop of adrenalin solution is applied, but returns as soon as the effect of the adrenalin has worn off, proving that it is caused by a vessel dilatation, and is not due to inflammatory products in the tissue between the network of small cutaneous vessels. It is quite evident that we must be dealing here with some profound systemic intoxication, and that it arises from the digestive tract is indicated by the fact that if one of these infants becomes ill with some intercurrent infection so that he will not eat, the skin condition clears like magic, and returns again as soon as he starts

*Moro's "Erythrodermia Desquamativa."

to take his food. We have seen this happen a number of times. We recall in particular one girl of four years who had had this condition ever since she was a small baby. During exacerbations I have seen erythematous patches even on her palms and soles. After an exacerbation the outer layers of the skin died and there was a good deal of peeling of dead skin, as in scarlet fever. This particular patient was in the Children's Hospital for many weeks, and was studied most carefully by every means at our command, including elimination diets, but we were never able to get at the cause of her trouble or do her any good. Skin tests to egg were always positive, and had been so ever since she was a baby, but omission of egg did no good. We have had several more such cases as this where we have never been able to do the patient the slightest good, and the bad results could not have been due to our own stupidity, for most of these bad cases had been to one or more good dermatologists before we saw them. We have had three similar cases with the same picture of intense generalized erythema, infiltration, and blue and cold hands and feet, where the condition has been cured entirely by means of a milk free diet, in spite of negative tests to milk protein.

Such cases represent the greatest intensity of the eczematous process, and the futility of hoping for cure by local treatment is quite apparent.

Their appearance is so characteristic that I think they would warrant inclusion in a special group if one were to attempt a classification, and it may be that we are dealing with an entirely different condition than the ordinary case of eczema, but it is possible that these dreadful cases simply represent the eczematous process greatly intensified. In none of these cases have we ever found by means of the skin test any clear-cut sensitization to any one specific food protein that helped us therapeutically.

At the other extreme is the fat, healthy infant with red cheeks. These cheeks are somewhat too red, and become very rough and irritated in the winter. Changes of temperature, water and soap aggravate the condition. This infant represents the mildest degree of the eczematous process, and he too has a capillary dilatation. He has it on the cheeks, because the capillaries there contract and dilate more easily than those on any other part of the body, and the cheeks are more exposed to external irritation. If internal irritation plays a rôle, and it probably does in all except the mildest cases, the cheeks are first and most severely affected for the same reason.

The food proteins which are most likely to give positive tests are those of egg, milk and wheat. We have in our present series 456 cases of infantile eczema, and have done skin tests on 132 of these, all relatively severe cases. Of 132 cases tested, 76, or 57 per cent gave positive tests, 39 per cent to egg, 15 per cent to milk, and 9 per cent to wheat. Of 52 cases which gave positive tests to egg, 30 had never eaten it, and they were not getting it through breast milk, because they were bottle-fed babies. If these cases are excluded, 47 per cent of 132 cases tested gave positive tests to something they were eating. So it may be said that the skin tests are of possible value in about half the cases. When one attempts to evaluate the benefit that is obtained by omitting foods which give positive tests, it is extremely difficult to arrive at an impartial and accurate judgment. We can arrive at only certain general conclusions. When multiple sensitization exists, it has seemed to us that withdrawal of all the supposedly offending foods is not very satisfactory, and is more likely to do harm to the child's nutrition than good to the eczema. Where very definite sensitization to one or two foods exists, and when the skin tests are supported by the clinical history, sometimes very striking results may be obtained, particularly with infants, taking an exclusive milk diet, who are sensitive to milk. So many eczematous infants and children react to egg, whether or not they have eaten it, that the importance of egg as an immediate cause can by no means be measured by the frequency of reactions, and yet it cannot be denied that in some cases removal of egg gives very prompt results. Usually, if a food is causing the eczema, it takes only a short time for the eczema to be cured when the food is omitted, but sometimes, particularly when there is sensitization to more than one food, improvement may be more gradual. It seems to us that in many cases positive food tests are of little practical value, that some reactions may not be specific at all, *and that there may be going on some underlying and much more complicated process of which the reactivity of the skin to food proteins is simply a part.* It cannot be denied, however, that the conception of food allergy has helped more than has any other one thing, and we should be very sorry indeed to be deprived of the skin tests, in spite of the fact that they are often contradictory and confusing.

Most of the eczematous infants that one sees are bottle-fed, and yet sometimes the most severe cases are seen in breast-fed infants. These babies are usually fat, and their eczema is almost always of the moist type. They are so fat, and improvement is so likely to follow reduction of their food supply by dilution with water before or after nursing, that it seems quite reasonable to assume that overfeeding has a good deal to do with their trouble. Whether it represents overfeeding with any single food element, or overfeeding in general, I do not know. In 1922, O'Keefe showed that about 60 per cent of 41 breast-fed infants gave positive tests to foods that the mother was eating, mostly egg and milk, but in some cases to almost any food. Occasionally tests would be positive to almost everything the mother was eating. This work was later confirmed and amplified by Shannon.

They both found that by omitting the offending foods from the mother's diet, the eczema was either cured or greatly improved. This was a decidedly new and original thought, and caused a very considerable interest. Its possibility has, however, been denied by a number of investigators. The most recent work is by Donnally of Washington, who showed quite definitely by means of improved technique that traces of egg may be found in the breast milk. Walzer also showed that absorption of unaltered protein is very common in even normal persons. The weight of evidence in the light of these newer investigations seems to be that it is possible for traces of foreign proteins to be present in breast milk. This by no means answers the question of their importance in causing eczema, and when one finds that a nursing infant may react to ten or a dozen foods that the mother may eat, all the way from egg to artichoke, he becomes a little skeptical about the specificity of the tests. Furthermore, one would expect that if such an infant, having given negative tests to milk proteins, but positive to several others, were weaned, and fed an exclusive cow's milk diet, he would be rapidly cured of his eczema. Such, however, is not likely to be the case. While most of our eczematous infants have been bottle-fed, we have, however, dealt with enough breast-fed ones to have reached the conclusion simply from clinical observation, that transmission of sensitizing material through the breast milk may take place in some cases, and we think that this aspect of the situation should be taken into account in dealing with the baby. We are very skeptical, however, about the validity of sensitization to a large number of foods, and we do not advocate a wholesale omission of foods from the mother's diet. As a matter of fact, if the eczema is a very severe one, we prefer to wean the baby, and put it on a milk-free diet.

Most eczematous infants are bottle-fed, and are taking no food but cow's milk into their digestive tracts, so if one assumes that the disease may have its origin in the food supply, he is immediately forced to the conclusion that something in cow's milk is the responsible factor. Blackfan and Schloss in their earlier work stressed the importance of this, and more recently Variot in France and Kerley here have looked upon cow's milk protein as the chief source of eczema in bottle-fed babies, and have advocated the prolonged heating of the milk in order to bring about changes in the protein, just *what* changes I do not know, to render it less likely to cause eczema. Variot, in particular, has been enthusiastic, and has written several papers on the subject in the last five years. There is enough in the literature, both foreign and American, to make one give serious attention to the rôle that milk may play, and a close study of this food in its relation to infantile eczema is of considerable importance in trying to get at its cause.

In our series, 15 per cent gave positive reactions to milk protein, with casein and lactalbumin about equally divided, and about half giving positive reactions to both. We have had in this series only four cases of very severe sensitization where the taking of small amounts of milk would immediately bring about an acute exacerbation of the eczema. We have seen a number of cases of true milk idiosyncrasy, where the infant was made violently ill by milk, but these cases do not as a rule have eczema, for the infant is not able to take enough milk to give it to him. In order to study the effect of milk, it is necessary to have a milk free food which the infant can digest and upon which he will thrive. Such a food is Sobee, made from Soy bean flour, homogenized olive oil, purified cornstarch, and various salts. We have fed 76 eczematous infants on this food.

It is always essential when one is interested in a therapeutic procedure, and wishes to see good results, to be unusually cautious in maintaining an impartial and critical attitude, and not be carried away by undue enthusiasm. This attitude we have tried to maintain.

In 13 cases for various reasons no definite conclusion could be reached. Twenty-six cases were entirely cured, and the cure was so much more striking and rapid than could have been accomplished by any other method of treatment that there can be no question that milk was the cause of the eczema in these cases, although only seven gave positive reactions to milk protein. Fourteen cases showed such definite improvement, without entire cure, that the withdrawal of milk could be considered a satisfactory procedure. Eleven cases would not take Sobee. Most of these were older infants, and it is always difficult to get infants a year old or over to take it. In ten cases, although they took the food well, digested it well and were kept on it for a considerable period of time, there was not the slightest improvement in the eczema. Two infants were unable to take it, because it made them vomit. Leaving out the cases from which no conclusion could be drawn, and those who would not take the food, it may be said that in about half of a series of 54 cases of infantile eczema, withdrawal of milk was followed by such striking and definite improvement that there seems no question that some property of milk was the cause of the eczema. What is the explanation of this? Only seven of the 26 cured cases gave positive reactions to milk protein. It is well known, however, that an individual may be sensitized to a protein, and still give negative skin tests to it. A possibility is that some of these patients may not have been sensitized to whole milk protein, but to some of the higher intermediate products

in the digestion of protein, such as albumoses and peptones, and we hope later to do skin tests with these products. It would not seem that the total amount of protein enters into the question, for Sobee as it is prepared for consumption contains even a larger amount than would most milk mixtures. It would not seem as though the fat, sugar or salts of milk could be blamed, as Sobee contains as much fat as does milk, although to be sure a different fat, and it is also rich in added salts. The answer may lie in the relative indigestibility of milk protein. Most bottle-fed infants are taking much larger amounts of protein than they need, and when one considers the difficulty of digesting milk protein, it would not be surprising that some of it might well escape complete digestion and be absorbed into the blood stream before it reaches the amino acid stage of digestion. It is not unreasonable to assume that some such process does take place, for Walzer has shown that the absorption of undigested protein is very common even in normal individuals.

We do not wish to give the impression that a milk free diet is a cure for most cases of eczema, for this is by no means so; it is merely one extra way of getting at the disease that may help, and I should recommend using it only in severe cases, for no milk free diet is so good a food for infants as milk, although as a matter of fact Sobee seems to fulfill the purposes of nutrition very well. Although there are so many gaps in our knowledge of infantile eczema, one is by no means helpless when it comes to the practical management of a case. Local treatment is of great importance, and when handled skillfully, helps a great deal. Occasionally brilliant results are obtained by a study of the allergic reactions, and regulation of the diet. As a routine food in the milder cases, I like to use either evaporated milk or Similac, and believe that in the long run most cases do better on either one of these two foods than they do on ordinary fresh milk mixtures. Sobee is reserved for the very severe cases or those having a definite milk sensitivity.

There is a great deal more to infantile eczema than scratching the skin, finding the offending food, omitting it from the diet, and curing the child. This formula has a fine sound, but is likely not to work. There is more to it than omitting milk from the diet. There is more to it than local treatment. What the missing factors are, I do not know.

FEE SPLITTING

This unethical and vicious practice is a perennial topic for discussion in addresses by officers of medical societies. It was referred to by Dr. Frank Lahey in his presidential address before the New England Surgical Society in the recent annual meeting at Hartford, Connecticut. He believes that it is not a very common practice in the eastern section of this country but is somewhat prevalent in the states west of the Atlantic seaboard.

In the code of ethics of the American Medical Association and that of the American College of Surgeons this practice is condemned vigorously. It has no standing in any reputable medical society and yet it exists if the general accusations are founded on facts.

For some unexplained reason a practice so generally condemned must exist on the theory that smoke is an indication of fire. If it exists to any great extent it is strange that so few doctors have been called before disciplinary boards. Even the alleged fee splitting by the brother of the recent Mayor of New York seems to have had more political significance than constructive attention by the Medical Society if we can interpret the sentiments expressed by an editorial in the *New York Times* of September 27 ult. in which Corporation Counsel Hilly is scored for his indifference in an investigation of four doctors which led to the question in the editorial: "Did the four physicians, in pure kindness, charity, admiration, divvy with the Mayor's brother?"

Mr. Hilly crawls from under his supposed duty by the alleged statement that it "is a matter for the medical profession to take up" and the grievance committee of the State Board of Medical Examiners in turn "feels that it may not, as a statutory body, determine that it is unethical" to split fees.

Here we are just as we were before this matter became notorious and we must wait for some pronouncement by the New York Board of Regents which, according to the *New York Times*, has the matter under advisement.

The mention and denunciation of fee splitting seem to be more of a gesture than a call for action, for it is likely that a real effort to get at the facts would be productive of good if medical societies could be induced to get the evidence. A good detective could secure the facts. Even though there may not be anything criminal in fee splitting a public announcement that Dr. Blank had been found to engage in this practice would at least appraise the public that he and his associates were engaged in a combination which is generally claimed to be contrary to the best interests of the patient. It would be of interest to see what would happen to a doctor's practice if his patients knew of his connivance with another in splitting fees.

Perhaps the inactivity of medical societies is the reason why so little notice has been taken of the commissions paid by supply stores to doctors for prescriptions or mechanical appliances furnished to patients. This unfair practice of diverting purchases of medicine, trusses or instruments to the fee splitting vendor of these articles is a matter that can be easily proved. Yet no apparently practical interest in either form of fee splitting has come to the surface. The patients are not the only sufferers, for the honorable vendor of medicines or mechanical appliances and the conscientious doctor are both the victims of an undercover competition which is difficult to meet. The profession can stand more firmly intrenched in public confidence if it will attack both of these irregular methods.

PULMONARY EMBOLISM FOLLOWING SIMPLE FRACTURE

BY FRANKLIN G. BALCH, JR., M.D.*

PULMONARY embolism following simple fracture is, fortunately, a rare occurrence. Henderson reported three cases from the Mayo Clinic following closed reduction of fractures on the long bones and one case following multiple fractures without any attempt at reduction. These cases were confirmed at autopsy. Cotton-Cornwall, Ponder, and Evans[2] reported a case of a woman of forty-three who received a simple fracture of the tibia and fibula. She was treated in a plaster cast. She died suddenly some six weeks later after getting out of bed. Autopsy showed a clot about the size of the internal iliac vein occluding both pulmonary arteries. The original source of the clot was not stated. Henderson felt that pulmonary embolism in general was apt to occur in elderly individuals who were overweight and who had subnormal blood pressure.

At the Massachusetts General Hospital from 1917 through 1931, there were but two cases of pulmonary embolism following simple fracture, though there were four or five times as many following compound fractures or amputations because of fractures.

A summary of these two cases follows:

J. C., a fifty-eight year old fisherman, fell from a wharf, a distance of some ten feet, sustaining a fracture of the neck of the right femur. He was brought to the hospital in an ambulance, a distance of one hundred miles, the same day. A plaster spica was applied on entrance. As the position was not satisfactory, a new spica was applied six days later. He suddenly died while lying in bed ten days later. Autopsy by the medical examiner showed "pulmonary embolism secondary to thrombosis of the iliac veins."

J. G., a twenty-eight year old salesman, was escaping from a fire over a rope made from sheets. This broke dropping him two stories. He sustained bilateral Colles' fractures. These were reduced under gas anesthesia. Nine days later, while up and about the ward, he suddenly complained of pain in the chest, became cyanotic, and died within a few minutes. There was no autopsy report on this patient.

A case which I saw recently is of interest and will be reported in some detail.

W. R., a retired laborer of sixty-one, was struck and knocked down by an automobile. Examination showed a man who looked his years and more. There was no evidence of a fractured skull. The left humerus showed a fracture of the mid third with angulation but no overriding. There was marked pain on the left side of the chest on respiration with subcutaneous crepitus below the nipple. There was some pain in the left leg near the knee. The heart showed a blowing systolic murmur at the apex. There was a left inguinal hernia. The x-ray showed a fracture of the eighth rib and a comminuted fracture of the humerus. The arm was put up in extension in a Thomas splint and the chest strapped with adhesive plaster. Nine days after admission he began to complain of discomfort at the left knee, though he could flex it normally. An x-ray taken then showed a fracture of the head of the left fibula "parallel to the long axis of the bone" and with "very little if any displacement". It was noted in the same x-ray that "both the anterior and posterior tibial vessels were calcified and also the vessels of the dorsum of the foot".

Two days later, about midnight, he suddenly became very dyspneic and cyanotic and died in about forty-five minutes.

Autopsy, performed by the medical examiner, showed fractures of the second to the twelfth ribs on the left, thrombosis of the left femoral vein up as high as Poupart's ligament, considerable aortitis, and multiple small pulmonary emboli.

In retrospect it is difficult to see what could have been done to prevent such an outcome. Stasis is thought to be a factor in the formation of thrombi, but in this case the leg was not immobilized at any time.

*Balch — Assistant Surgeon to Out-Patients, Massachusetts General Hospital. For record and address of author see "This Week's Issue," page 679.

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PIGMENTATION FROM PERFUME

"Berlock" Dermatitis

BY J. G. DOWNING, M.D.†

THE use of cosmetics and perfumery is becoming increasingly widespread, not only among women, but among men. One of the largest industries today is the so-called beauty culture business.

Occasionally, as a result of these many applications, the physician is presented with a disturbance of the skin. The most common result is a persistent discoloration, or pigmentation. These skin discolorations have been, at times, attributed to some organic disease, their true nature not having been recognized, and the patients submitted to the discomfort of routine physical examinations. Discoloration of the skin is frequently seen in a dermatological practice.

Perfumes and toilet waters will, in some cases, produce a dermatitis. However, their

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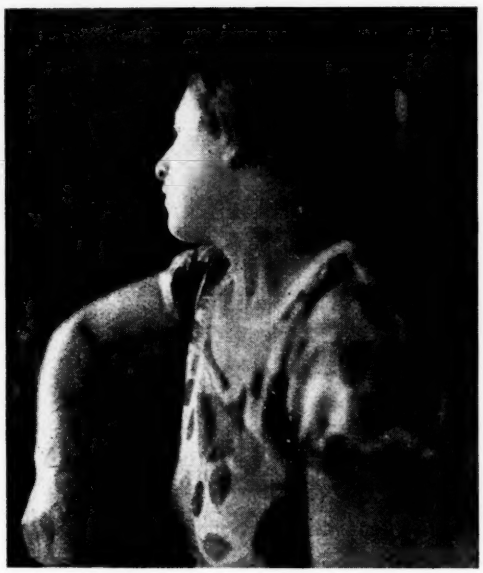
number and variety is so infinite that it is rare that one finds any group of cases showing the same result from one definite substance. Therefore, it has been deemed worth while to report three cases in which pigmentation resulted from the use of the same brand of perfume, followed immediately by exposure to sunlight:

On June 9, 1930, Miss C. C., a telephone operator, reported to my office, complaining that the ear phones had produced a discoloration of the skin of the neck. Questioning revealed that she had recently been presented with a bottle of French perfume, Shalimar, made by the Guerlain Company, and that she had applied it to her neck just before going canoeing. Examination of the skin showed a semicircular area below the right ear, one and a half inches in diameter, a similar semicircle just below, and a third area, pear-shaped, with the smaller part of the oval at the lower border over the center of the sternomastoid muscle. These lesions were of a brownish-yellow color.



CASE 2.

Showing Typical Area of Pigmentation.

On June 10, 1931, Miss D. G. reported to me, complaining that three days previously a brown spot appeared on the left side of her neck, accompanied by swelling of the lobe of her left ear. She stated that she had applied a French perfume called Shalimar, by means of the glass stopper of the perfume bottle, to both of these areas before sitting in the sunlight. She showed on the left side of her neck, just below the ear, an area of light brown pigmentation, Indian club shaped, with the tip pointing downward. The lobe of the ear was intensely red, edematous, and covered with fine vesicles.

Mrs. A. D., on February 19, 1932, while in Bermuda, noticed dark brown streaks on the skin of both sides of her neck. It was her habit to stroke the sides of her neck with the long glass applicator of her perfume bottle of Shalimar before going outdoors. She showed a wide streak of brown pigmentation on both sides of the neck, definitely demarcated, and darker at the bases than at the upper portions.

The first two patients were not seen a second time. The first was given no treatment. A soothing lotion was applied to the lobe of the ear of the second. The third patient was given the following desquamating lotion:

Corrosive Sublimate	0.25
Tincture of Benzoin	3.0
Zinc Sulphate	1.0
Alcohol	25
Distilled Water	25

Following this application the pigmentation disappeared in about three weeks.

To test the effect of this perfume, it was applied to a very blond skin, a brunet skin, and the skin of the last patient. After it had dried, these skins were exposed to the radiation of mercury vapor arc. An erythema dose was given in each case, with resulting erythema, which appeared except where the perfume had been applied. Here the skin remained normal in color in each case, showing that the perfume acted as a filter.

The dermatitis of the lobe of the ear in the second case would agree with Highman's statement made in the discussion of the paper of Lane and Strauss[1], that he would place perfume dermatitis in the group of dermatitis venenata. At the same time he reported a male patient who suffered a peculiar dermatitis of his face which later was found to be due to a French perfume which his wife had been using, showing the wife could be the intermediary host.

The type of pigmentation reported in the above cases has been known for many years. In 1916 Freund[2] reported four cases observed in 1914 which showed a peculiar pigmentation of the skin with Eau de Cologne, followed immediately by exposure to sunlight. He made experiments which prove the Oil of Bergamot was the offending ingredient of the Eau de Cologne.

Eight years later Franz Rosenthal[3] presented a case in Berlin and named the eruption "Berlock" Dermatitis because the streaks of pigment reminded him of the pendant of a necklace or watch chain. At this time he did not recognize the cause, but thought it was due to bacteria. In the same year Hoffmann and Schmitz[4] proved the cause of the condition and also established the fact that Freund was the first to demonstrate the condition and its etiology.

In 1927 Cole's[5] investigation of cosmetics by a questionnaire sent out to American dermatologists revealed four reports of dermatitis due to toilet water. The favorite areas for the application of these seductive agents seem to be behind the ears, on the neck, the axillary fossa, and the breasts, although Pozzo[6] reported one case occurring on the arm and Langer[7] and Samek[8] each reported a case on the cheek.

The cause of the eruption in most of the cases first reported was Eau de Cologne, although Foresti Brito[9] reported a case following the use of Birkenwasser. Gross and Robinson[10] in New York reported five cases due to one brand

or another of a well-known imported perfume, designated as G. M. and G. S. Lane and Strauss[1] reported a case due to Bervaine, a preparation which is known to contain Oil of Bergamot. Strauss stated in the discussion of his paper that susceptibility to Bergamot is a necessity. As in the second case reported above, various degrees of dermatitis have been reported by Galewsky[11], Linser[12], Falkenstein[13], and Freund[14].

Excessive perspiration has been considered a factor in the production, according to Zurhelle[15], who stated that blonds showed a greater tendency toward dermatitis, while brunettes showed more pigmentation. Attempts to produce pigmentation by such methods in cases of leukoderma have been reported by various writers, some claiming considerable success, and others failure. Axmann[16] reported that he produced pigmentation in leukoderma after rubbing in Eau de Cologne. Uhlmann[17] and Szanto[18] also reported a series of successful cases, while Bonnet[19] and Goodman[20] reported entirely negative results. The difference may be due to the type of Oil of Bergamot used by the various experimenters, for it is impossible to find out the exact ingredients in an offending substance. Each industry, and especially the perfume industry, has developed special technique which is usually considered as a part of the body of "trade secrets" known only to an interested few. There is no doubt that much more is known about Oil of Bergamot in the perfume industry than is available in the ordinary references.

Oil of Bergamot is obtained by expression of the fresh peel of the ripe fruit citrus bergamia[21]. Within limits, the value of the oil which is chiefly used for perfumes depends on the esters it contains. For this reason, it is important to bear in mind that the presence of fatty oils and resins will increase the apparent content of esters, even though this oil is thus adulterated. Besides adulteration with fatty oil and resins, Bergamot Oil suffers from the addition of oils of turpentine, lemon, orange, and distilled Bergamot Oil, which is obtained by rasping the peel of fallen fruit and distilling the product with water. Another adulterant is prepared by treating oil of turpentine with hydrochloric gas. Dermatitis resulting from such applications may not be due to the natural Oil of Bergamot, but to its adulterations.

CONCLUSIONS

1. Three cases of pigmentation are reported due to the combination of perfume and sunlight.

2. Artificial light and the same perfume did not produce a pigmentation on a person previously affected by the same combination. It seems, therefore, that a source of artificial light cannot produce the same results that noonday sunshine will.

3. Oil of Bergamot, which is chiefly used for perfumery, is the offending substance in most cases reported in the literature.

4. Pigmentation thus caused is transient and can be removed by a simple desquamating lotion.

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SMALLPOX IN UTERO

BY BENJAMIN G. HORNING, M.D.*

SMALLPOX in utero is sometimes observed and a number of cases are recorded in the literature. The subject of this report resides in Fairfield, Connecticut, where an outbreak of smallpox had recently occurred.

Mrs. J. S. is a French-Canadian housewife of forty-one years. She was married at the age of nineteen and has been delivered of nine full term, healthy children. There have been no miscarriages or stillbirths. There was no history of venereal disease and the Wassermann test was negative. She had not previously been exposed to, or vaccinated against smallpox. Otherwise the past and family history was not important.

This woman had the onset of an illness on March 3, 1932, diagnosed as a typical case of smallpox from which she made an uneventful recovery. She was in the sixth month of her ninth pregnancy at the time. Her child, a normally developed and well-nourished son was born on June 9, 1932, ninety-eight days after the onset of the mother's illness. There were many healed lesions on the surface of the infant's body characteristic in distribution and appearance of smallpox. Apparently desquamation had been delayed while the body was suspended in the amniotic fluid but proceeded rapidly when exposed to the air.

SMALLPOX IN UTERO.

Baby S. Born Thursday, June 9, 1932, at 11:55 P. M. Pictures taken Saturday, June 11, 1932, at about 3 P. M. Mother had smallpox with onset March 3, 1932.

The diagnosis was smallpox in utero. The patient had been infected, had the disease and recovered before birth. The accompanying pictures were taken about forty hours after the child was born.

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THE CROSS-EYED CHILD

The National Society for the Prevention of Blindness has issued a strong appeal for the early correction of strabismus with the suggestion that the family physician must correct the erroneous opinion that treatment should be delayed until the child has lived more than four years, thus giving indications of established good health.

The danger of imposing depressing handicaps on a young child by unfeeling remarks of his playmates and the growing appreciation of his disability react definitely on the mentality of a sensitive nature which may not only retard self-expression but lead to distinctly abnormal traits.

The child with a squint should be taken in hand as early as two years and put under the care of a competent adviser and not left to "outgrow" the deformity as is sometimes advised.

This, as well as any other obstacle to good physical and mental health, must be overcome if the child is to have the best possible chance of attaining success in later life.

MORTALITY RATES

Telegraphic returns from 85 cities with a total population of thirty-seven million for the week ending Oct. 1, indicate a mortality rate of 9.4 as against a rate of 9.6 for the corresponding week of last year. The highest rate (16.1) appears for Spokane, Wash., and the lowest (4.3) for Flint, Mich. The highest infant mortality rate (11.4) appears for Tampa, Fla., and the lowest for Bridgeport, Conn., Des Moines, Iowa, Fall River, Mass., Lynn, Mass., and Worcester, Mass., which reported no infant mortality.

The annual rate for 85 cities is 11.2 for the thirty-nine weeks of 1932, as against a rate of 12.0 for the corresponding period of the previous year.—*Bureau of the Census.*

MEDICAL PROGRESS

ADVANCES IN NEUROLOGICAL SURGERY

BY TRACY J. PUTNAM, M.D.*

BRAIN TUMORS

A CONCISE review of the subject of brain tumors by Harvey Cushing (Intracranial Tumours: Baltimore, C. C. Thomas. p. 150) leaves very little to be said upon the subject. This little book contains a review of two thousand verified neoplasms from the author's clinic. They are arranged on a pathological basis, with illustrative case histories and a computation of the surgical mortality for each group. The book is profusely illustrated and written in a style which makes it difficult to put down before it is finished—a masterpiece of medical writing.

An extensive critical review of the same subject is given by Walshe (*Quart. J. Med.* 24:587). Here the material is drawn almost entirely from the literature. A valuable bibliography is given.

Elsberg has continued his series of papers on the subject of meningiomas in various locations —the parasagittal tumors (*Bull. Neur. Inst.* 1:389) and those on the under side of the temporal lobe (ibid. 2:95). He emphasizes the necessity of understanding in each case the blood-supply of the tumor, and gives his methods of dealing with each in some detail. The papers are particularly to be recommended for their precise technical directions.

Kant (*Arch. f. Psychiat.* 93:343) calls attention again to the fact that a "paretic" gold curve may be caused by tumor. There are other important changes in the spinal fluid in cases of tumor on which great dependence is placed in this clinic. A paper on the subject is now in preparation.

A simple, practical classification of gliomas is presented by Penfield (*Arch. Neurol. & Psychiat.* 26:745). It is more easily grasped than that of Bailey but it should be remembered that the latter's work is based on a larger material.

According to Cairns and Russell (*Brain,* 54:377) not only medulloblastomas but also astrocytomas, ependynomas and glioblastomas may metastasize through the cerebrospinal fluid spaces.

Hamburger (*Am. J. M. Sc.* 181:756) discusses the various possible types of head murmurs. Those of intracranial origin include some apparently not due to structural abnormality—the "head murmurs" of children, often associated with anemia. Arteriovenous aneurysm and vascular tumors are a familiar source of murmurs. In one instance, angulation of the vertebral artery from a cerebellar tumor evoked a murmur. Bruits may also be transmitted from aneurysm or atheromatous segments of the carotids.

Marked intellectual impairment, drowsiness, apathy and emotional lability were found in three adult patients with posterior fossa tumors, reported by Southerland (*Bull. Neur. Inst.* 2:144). On the other hand five out of 50 patients with frontal lobe tumors showed such marked cerebellar signs that the diagnosis of tumor of the posterior fossa was considered (Hare: *Bull. Neur. Inst.* 1:532). In all five instances the tumor was bilateral.

Güttich (*Monatschi. f. Ohrenh.* 65:272) reports a successful extirpation of an acoustic tumor by the translabyrinthine route and there are others on record. The mortality, chiefly from meningitis, is shockingly high, however. Cushing's new method of "uncapping" the tumor by resection of the cerebellum makes such procedures obsolete.

In two cases of cerebellar hemangioblastoma reported by Davison, Schick and Goodhart (*Arch. Neurol. and Psychiat.* 25:783) peculiar disseminated degenerative lesions of the cord coexisted.

The retina will tolerate radon seeds of at least 5.3 millicuries, according to Moore, Stallard and Milner (*Brit. J. Ophth.* 15:673) and this amount of radiation will cause the regression of some retinal gliomas. Perhaps this would be worth trying with gliomas of the optic nerve also.

From a study of seven cases of tumor of the basal ganglia, Ody was unable to formulate a definite syndrome (*Arch. Neurol. & Psychiat.* 27:249). In several instances there were manifestations suggestive of decerebration, but dystonia, tremor, rigidity, thalamic pain and other symptoms which might be expected were almost wholly lacking.

TUMORS OF THE PITUITARY REGION

A new syndrome apparently due to basophil tumor of the anterior lobe of the hypophysis has been described in a brilliant paper by Cushing (*Bull. Johns Hopkins Hosp.* 50:137). There is acute plethoric adiposity, genital dystrophy, vascular hypertension and atrophic striae of the skin. Some of the patients have been benefited by x-ray treatment of the hypophysis.

An important review of the surgical approach to tumors in the region of the optic chiasm is given by Heuer (*Surg. Gynec. Obst.* 53:489). After a thorough historical account of the de-

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velopment of the various operations which have been suggested, he discusses their relative value. He summarizes not only his own experience but that obtained by personal communication from other members of the Society of Neurological Surgeons. The transsphenoidal operation appears to have been largely abandoned in this country as it gives a much less adequate exposure than the suprachiasmal procedures and the mortality is about the same in both instances. The article is beautifully illustrated but no new cases are reported.

The results of a large and successful experience with tumors of the hypophysis are concisely given by Hirsch (*Klin. Monatsbl. f. Angenh.* 85:609). The method which he has developed is a modification of Schloffer's transnasal approach. Following the operation he leaves a plaque of radium against the ethmoid, supported by a rod held in position by a dental plate. In 103 cases there was an operative mortality of 4.8 per cent. Of 25 patients followed from four to ten years since operation, 22 have retained their improvement (usually very marked). This is an enviable record. It is to be objected, however, that the mortality is about the same for the transfrontal operation in skilled hands and that x-rays persistently applied should yield as good results as radium. The exposure is better by the transfrontal route and other lesions encountered unexpectedly may be dealt with. The pathology of lesions of the hypophysis is well described. In another paper Hirsch and Kotrnetz (*Arch. f. klin. Chir.* 168:85) describe the variations in topographical relationship between the chiasm and the hypophysis.

Pfahler and Spackman (*Am. J. Ophth.* 14:796) report some excellent results from x-ray treatment of pituitary adenomas. The chief manifestation of improvement has been return of vision. Other neighborhood signs have also regressed and the skeletal changes of acromegaly have been arrested. They believe that the tumors which fail to respond to radiation are cystic and that these are the very ones which are most apt to yield brilliant surgical results. They report improvement in one case of meningioma of the sella—an astonishing result. They recommend high voltage radiation through several small portals. The positive results recorded are striking but the authors appear unduly pessimistic as to the possibilities offered by surgery.

Essentially similar results from radiotherapy are reported by Dyke and Gross (*Bull. Neur. Inst.* 1:211).

Frazier (*Surg. Gynec. Obst.* 52:1069) reviews 334 cases of pituitary disease in his collection. His findings and conclusions are too extensive to be reported here. He advocates radiation for mild pressure symptoms and transfrontal operation if there is severe visual loss. The operative mortality is about 5 per cent.

A valuable paper on the x-ray diagnosis of tumors in and about the sella is contributed by Pancoast (*Am. J. Roentgenol.* 27:697). A detailed account of the anatomy of the region is given. No dependence can be placed on measurements of size unless a standardized technique is used. Abnormal vascularity may cause atrophy of the clinoids. It may be possible to make the diagnosis of tumor from enlargement of the sella alone but absence of enlargement does not exclude tumor.

De Martel, Denet and Guillaume (*J. de chir.* 37:321) give a minute description of the technique of the transfrontal operation. Their method follows closely that employed in this country.

An obstructive hydrocephalus, apparently the result of a previous meningitis, caused distention of the sella and bitemporal defects in a patient described by Sinclair and Dott (*Tr. Ophth. Soc. v. Kingdom* 51:232).

A unique case of hemorrhage into a preëxisting pituitary tumor following trauma is reported by Van Wagenen (*Ann. Surg.* 95:628).

SURGERY OF THE SPINAL CORD

An important paper by Kernohan, Woltman and Adson (*Arch. Neurol. & Psychiat.* 25:679) describes the histologic types of 51 cases of intramedullary tumor of the spinal cord. The average duration of symptoms was 4 and 9/10 years which is longer than the average duration of extramedullary tumors. In two instances choked discs were found and in one of these quadrantanopsias. (It would be interesting to hear more of this case.) Four instances of extramedullary ectopic gliomas were encountered.

The types were as follows:

Ependymal tumors	21
Spongioblastomas, varia	7
Unipolar spongioblastomas	2
Spongioblastomas multiforme	4
Astroblastomas	2
Oligodendrogliomas	2
Medulloblastomas	4
Ganglioneuromas	1
Hemangioblastomas	4
Fibroblastomas	1
Lipomas	1
Tuberculomas	1

Of nine cases in which the entire cord was available for study, syringomyelia coexisted in five.

In a brief article, Craig (*Am. J. Surg.* 12:303) gives the statistics of 312 cases of cord compression from the Mayo Clinic. Of these, 223 were due to tumors outside the cord, mainly endotheliomas and neurofibromas. The intramedullary tumors were mainly gliomas but in some enucleable benign tumors were found and other cases were benefited by decompressive laminectomy.

Cairns and Riddoch (*Brain* 54:117) report two cases of ependymal glioma of the cord in

which the tumor could be completely removed at one session. They advise more radical attempts to deal with intramedullary lesions.

A series of ventral chondromas of the spinal canal are reported by Elsberg (*Bull. Neur. Inst.* 1:350). The symptoms are usually vague and long-continued. They consist mainly of motor, rarely sensory loss, without disturbance in position or vibration sense. Sphincter disturbance is uncommon. There is usually little evidence of spinal block and x-rays are negative. They should be removed by a transdural approach.

VENTRICULOGRAPHY

A conservative estimate of the value of ventriculography is given by Adson (*Am. J. Roentgenol.* 27:657). He performs the puncture in the face-down position over the posterior horns. Both ventricles are tapped at once and air drawn in one needle by aspirating the other. (We have not succeeded in getting good filling by this method.) A valuable feature of the paper is an extensive tabulation of results. Ventriculograms were performed in forty-three cases with three deaths. The procedure led to a disclosure of the lesion in fourteen cases and was misleading in an equal number. Of course, it was only employed in cases in which the diagnosis was otherwise impossible.

Precise directions for an accurate routine for ventriculography are given by Deery (*Bull. Neur. Inst.* 1:193). The incisions are made transversely, 7 cm. above the external protuberance and 3 cm. on either side of the midline, with the patient in a semi-sitting position. Fluid is allowed to escape through one needle while air enters through the other. By using accurate measurements, deviations in position and variations in size of the ventricles can be accurately estimated.

Waggoner and Clark (*Am. J. Roentgenol.* 25:533) suggest that in taking encephalograms, the patient's head be flexed forward as far as possible and the upper part of the forehead pressed against the Bucky screen and casette. The tube is pointed at the occipital protuberance. The result is that the ventricles are seen almost as if from above which shows the third and fourth ventricles much more distinctly than does the conventional technique.

HEAD INJURIES

In a brief but meaty article (*J. A. M. A.* 98:1352) Kennedy and Wortis summarize an extensive article which is to appear (*Surg. Gynec. Obst.*) on the treatment and appraisal of head injuries. They propose the following criteria of structural neural injury:

(a) Absolute criteria.

1. Roentgen evidence of skull fracture.
2. Bloody spinal fluid.
3. Bleeding or cerebrospinal fluid leakage from orifices, especially from the ears.
4. Focal cerebral palsies.

(b) Presumptive criteria in the order of their importance.

5. Convulsive states, *proved* posttraumatic.
6. Ventricular distortion, *proved* posttraumatic.
7. History of prolonged unconsciousness.
8. History of adequate trauma.

It is hard to see why increased intracranial pressure by spinal manometry is not on this list. "All these units are really measurable and are instrumental in establishing the fact of brain injury."

They outline the usual treatment by lumbar drainage and intravenous glucose when shock has passed off. They recommend elevating the head of the bed and administration of 0.5 gm. caffein sodiobenzoate hypodermically every four hours. Operative procedures should be limited to compound and depressed fractures. They still believe in the efficacy of subtemporal decompression when coma does not respond to these measures after three hours.

A review of the symptomatology and extensive series of cases of head injury is given by Glaser and Schafer (*J. A. M. A.* 98:272). Organic signs were elicited in about 32 per cent, most of which were fractures of the base. If signs and symptoms lasted more than a year, the ultimate prognosis was poor. Only 9 per cent of patients who did not lose consciousness had persistent signs and symptoms as against 25 per cent of those who did lose consciousness. Convulsive attacks of undoubted organic origin occurred usually at least eight months after injury. A useful feature of the article is a series of tables and charts of the incidence of various manifestations.

Conservatism in the treatment of skull fractures involving the frontal sinus is urged by Gurdjian and Shawan (*Ann. Surg.* 95:27). Only compound fractures demand debridement. In 125 cases, the incidence of meningitis was 0.8 per cent.

According to Gurdjian and Schlafer (*Arch. Neur. and Psych.* 26:583), laceration of the orbital surface of the frontal lobe is common in cases of fracture of the skull with injury of the brain and is usually fatal. Extreme stupor, jacksonian epilepsy or paralytic changes of the face and possibly the upper limb, fracture in the posterior fossa, the vault or the anterior fossa and bloody spinal fluid were characteristic findings.

Leriche (*Presse méd.* 39:945) again calls attention to the possibility of a cerebrospinal hypotension following trauma to the head. He treats the condition by injecting 20-40 cc. of distilled water intravenously, then 500 cc. of ordinary salt solution. This may have to be repeated. The results should be checked by lumbar puncture.

Kaplan (*Brain* 54:430) has published a careful study of subdural hematoma. The diagnosis

is difficult in spite of the fact that the condition is more common than middle meningeal hemorrhage. The usual picture is that of alternate drowsiness and delirium coming on several days after a mild trauma. Homolateral paralysis is not uncommon. Dilation of the pupil on the same side as the lesion appears to be an important diagnostic sign. The lesion is often bilateral and both sides should be tapped.

Another paper by Fleming and Jones (*Surg. Gynec. Obst.* 54:81) deals chiefly with the surgical treatment of subdural hematoma. Of their eight cases, three had bilateral lesions. In one case, shift of the pineal revealed the side of the lesion. In all of these cases the hematoma was evacuated through two burr holes on each side, frontal and parietal, so situated that they could be employed in making a boneflap later if necessary, but all the patients except one (who was moribund on admission) were permanently relieved of their symptoms.

This is rather unusual. Temporary relief has often resulted from simple tapping but the symptoms frequently recur.

INFECTIONS OF THE NERVOUS SYSTEM AND MENINGES

Symonds (*Brit. M. J.* 1:45) calls attention to the diagnostic difficulties sometimes presented by the presence of increased intracranial pressure without intrathecal suppuration in cases of otitis media. Choked discs may even occur. The fluid is under high pressure and contains leucocytes but is sterile and contains a normal amount of sugar. The condition is remedied by draining the ear and withdrawing fluid.

Appelbaum (*J. A. M. A.* 98:1255) adds three cases of recovery from streptococcus meningitis to the forty-six already on record. Antistreptococcus serum was administered to two of these but the author emphasizes the fact that in his cases, as in most of the others, persistent drainage was the most important factor leading to recovery.

Three cases of spinal epidural abscess are reported by Allen and Kahn (*J. A. M. A.* 98:875). In only one instance could a definite source of infection be found. The diagnosis was made and operation performed in all three cases, but only one patient recovered, and then with a residual myelitis. Typical symptoms are an abrupt onset of pain in the back with fever, root pains, and signs of a transverse lesion.

An interesting collection of cases of cervical hypertrophic pachymeningitis is reported by Kment and Salus (*Beitr. z. klin. Chir.* 154:191). In six of 17 cases, the condition was luetic, in three tuberculous, in three metastatic, in three the result of a previous meningitis and in two the result of trauma. The symptoms were local pain, a sensory level, paraplegia, leucocytosis of the fluid, and arachnoid block. In practically all cases, operation proved necessary.

PERIPHERAL NERVES

An important and authoritative review of peripheral nerve injuries and their treatment is being run in the *American Journal of Surgery* by Pollock and Davis, beginning January, 1932.

A superb article on the operative treatment of facial palsy, written in collaboration with Dr. Arthur Duel, crowns thirty-seven years' work in this field by Dr. Charles Ballance (*Arch. Otolaryng.* 15:1). The authors review at length the various anastomoses proposed for facial palsy and conclude that the glossopharyngeal-facial is the most successful of them. They prefer, however, an intratemporal implant of a fresh graft taken from another nerve. Only one case is reported but a most convincing series of careful experiments on the baboon is given. They also recommend early decompression and inspection of the facial in all traumatic palsies and selected cases of the "rheumatic" type. The article is not only a valuable original contribution and compendium of available information but a charming personal document recalling vividly Sir Charles' imposing presence.

A case of torticollis which was not relieved by section of cervical posterior roots is reported by Dowman (*Surg. Gynec. Obst.* 53:836). The first four anterior roots as well as the accessory had to be divided also. The patient retained the ability to move his neck in spite of these extensive neurectomies.

Olivecrona (*Arch f. klin. Chir.* 167:293) has had excellent results from section of both anterior and posterior cervical roots, combined with extradural section of the spinal accessory, in torticollis. He warns against the use of this method in cases of striatal disease, but I have seen excellent results in such cases also when combined with my operative section of the reticulospinal tracts.

A simple though novel method of treating painful conditions in the extremities has been proposed by Dogliotti (*Presse méd.* 39:1249). With the patient lying in such a position that the affected roots are uppermost, he injects 0.4 cc. of absolute alcohol into the subarachnoid space. This floats on the spinal fluid and surrounds the offending roots which it attacks effectively. By the time it has diffused further it is so diluted that it has no further effect. He has worked out the technique on dogs. He reports a large series of cases, mostly of neuralgia and malignant disease. There is seldom any demonstrable sensory loss. The injection may have to be repeated. We have used this method successfully in three cases.

The use of sacral epidural injections for the relief of sciatica is urged by Lindemulder (*J. Nerv. & Ment. Dis.* 74:265).

For various reasons, Van Wagenen found it expedient to cut the trigeminal root in the posterior fossa in five cases (*Ann. Surg.* 94:1013). The operation proved sometimes easy, sometimes

very difficult. Complete anesthesia resulted when the root was completely divided. When the division was only partial, there was preservation of sensation over most of the face.

A surgical treatment of blepharospasm has been worked out by Frazier (*Ann. Surg.* 93: 1121). The number of nerve filaments to be cut may vary. The entire anterior edge of the parotid should be exposed and the individual tiny nerve filaments picked up individually between the superficial fascia and the muscle sheaths. They should be identified by stimulation (which precludes the use of a local anesthetic) and cut, not injected with alcohol.

SYRINGOMYELIA

Aside from the article from our clinic last year in this *Journal*, no further extensive articles have appeared. A short note by Peiper (*Arch. f. klin. Chir.* 167:318) reports a few brilliant successes and some dismal failures. We have personally not seen the aggravation of symptoms and difficulties in wound healing with this operation described by many authors although it must be admitted that no relief may be obtained.

Elsberg (*Tumors of the Spinal Cord*, New York, Paul B. Hoeber, 1916) has the undoubted distinction of being the first to report and recommend drainage of syringomyelic cavities. The operation should bear his name and not Puusepp's.

MISCELLANEOUS

The whole subject of chordotomy is treated in detail by Foerster and Gagel (*Ztschr. f.d.ges. Neurol. u. Psychiat.* 138:1). Twenty-nine cases are reported at some length. Two patients died of postoperative complications and, for the first time, anatomical studies of degeneration are given. In five cases there was no definite relief. One of these was a case of paralysis agitans—a most interesting report from the physiological point of view. Complete or partial relief was obtained in the remainder. Only occasional slight motor loss resulted. In some instances, there was a transitory retention of urine, and ileus. There is interference with sweating in cases in which the section reaches to the region of the pyramidal tract. Other important and instructive details should be consulted in the original.

The operative results in twelve cases of spina bifida are reported by Coughlin (*Ann. Surg.* 94:982). One patient died on the fourteenth day. In eight cases there was a spinal fluid fistula before operation. Only one child developed hydrocephalus. Paralyses were practically unchanged and the author believes that sphincter paralyses constitute a contraindication to operation. Details of operative technique should be read in the original.

A most valuable paper on the causes and treatment of jacksonian epilepsy is contributed by E. F. Fincher and the late Dr. Charles Dowman, whose tragic death is such a loss to the profession (*J. A. M. A.* 97:1375). The etiologic factors were as follows: brain tumor 24.6 per cent, trauma occurring after birth 20 per cent, birth trauma 14.6 per cent, post infection 10.7 per cent, cerebral atrophy 5.2 per cent, syphilis 5.3 per cent, arteriosclerosis 3 per cent, miscellaneous causes 3.8 per cent, undetermined causes 12.2 per cent.

Sixty-two cases were operated upon. The results were gratifying in the tumor cases, and encouraging even in the cases in which no gross lesion was enclosed. In the latter instance the "epileptogenous" area was injected with alcohol or coagulated.

A review of the surgical treatment of epilepsy is given by Guttmann (*Ztschr. f. d. Neurol. and Psychiat.* 136:1). The chief interest of the article consists in a report on the therapeutic results of encephalography and the excision of cortical scars. One case is specifically reported in which attacks ceased for a year after encephalography and in general the author states that improvement occurs in many cases. Often the improvement consists in a reduction of the necessary dose of luminal or bromides. He reports eight cases in which cortical scars were excised. None were permanently relieved of attacks but all were improved in one way or another. He very justly emphasizes the need of medical after-treatment. Of the catastrophes which occasionally follow the exploration of epileptic brains, he reports only one. He also makes no mention of negative explorations.

In two cases of migraine, Dandy (*Bull. Johns Hopkins Hosp.* 48:357) removed the inferior cervical and first thoracic ganglia with relief of pain. In the second case, removal of the inferior cervical ganglion alone was not sufficient and the first thoracic had to be removed also.

In last year's review, notice was taken of the work of Guleke, who repeated Dandy's classical work on obstruction of the aqueduct and found that not all animals developed hydrocephalus. His work has been repeated and confirmed by Heidrich (*Beitr. z. klin. Chir.* 151:607), who has reason to believe that absorption may occur from the ventricular walls and intercolumnar tubercle, especially in lower animals. He reports one human case, in which encephalography and injection of iodides showed a closure of the aqueduct, confirmed by necropsy, without hydrocephalus. Perhaps our views on the circulation of the spinal fluid should be more elastic.

Muscle may be preserved for several days in a tight container in the ice-chest, according to Davidoff (*Bull. Neur. Inst.* 1:343). In this clinic we have employed fresh pigeon muscle as suggested by de Martel, with great satisfaction.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 18411*

SUDDEN ONSET OF COUGH, FEVER AND BROWNISH SPUTUM IN A CASE OF CHRONIC DYSPNEA

MEDICAL DEPARTMENT

PRESENTATION OF CASE

DR. ALFRED O. LUDWIG**: This is the case of an eighteen year old boy who came into the hospital complaining of cough, fever and anorexia of three days' duration.

Seven days before he came into the hospital he developed nausea and vomiting. Three days before entry he developed cough and a high fever. He gradually grew worse and became dyspneic. For three days before entry he had been raising considerable amounts of brownish sputum. He had had 30 grains of digitalis in the three days before he entered the hospital. There were no chills or chest pain.

The family history is irrelevant except that his father complained of heart trouble.

The past history showed that since the age of seven the patient had had severe dyspnea on exertion. Three or four years before entry he had an attack of acute rheumatic fever with painful swollen joints. Four weeks before entry he had "grippe", with several severe night sweats.

Physical examination showed a fairly well developed and nourished boy with extreme cyanosis of the lips and fingers. He was in marked respiratory distress and sweating profusely. The heart was grossly enlarged to the right and left. There was a well marked systolic thrill at the base over the aortic area, also a loud harsh systolic murmur and a blowing diastolic murmur at the same area. No definite apical murmurs were made out because of the rapid rate at that time. The pulmonic second sound was accentuated. The aortic second sound was absent. There was Corrigan pulse, and capillary pulse and pistol shot over the brachials.

Laboratory studies showed a red blood cell count of 5.3 million and a white count of 28,850, hemoglobin 70 per cent, polynuclears 80 per cent, 16 per cent lymphocytes, 4 per cent mononuclears.

*The following report gives the essentials of two discussions, one in Dr. Cabot's Third Year class, one at a conference of the hospital staff.

**Recently senior interne on the East Medical service. In his absence in Europe it has been necessary to publish Dr. Ludwig's presentation of the case without his revision.

At entry the patient seemed moribund and was therefore immediately bled. With venesection, morphine and oxygen he improved very markedly. The following morning he had improved considerably and by that time showed definite dullness at both bases with bronchial breathing and egophony, with increased tactile fremitus at the right base. The left border of the heart at that time was 15 centimeters out in the sixth space. Mitral diastolic murmurs and systolic murmurs were well heard. The third morning he was quite dyspneic, cyanotic and uncomfortable because the oxygen supply ran out for a while. The fourth day he was somewhat delirious. Bronchial breathing and crepitant râles were heard at the left base also. X-ray showed a bilateral basal process. The temperature was 104°. The blood pressure and pulse had both risen. On the fifth day the temperature, pulse and respiration had gone down and he seemed better generally. The signs in the lungs were the same. Two days later he was delirious, but during the day he gradually improved; the respirations were quieter and the general condition better towards night. He continued to improve until about ten days after entry, when he suddenly had an attack of acute severe dyspnea and was delirious. The pulse and respiration went up rapidly and the pulse became very poor in quality. He had morphine, caffein and adrenalin, none of which seemed to benefit him. Then he had some coramin. That did not seem to help him either. He coughed up about two ounces of bright bloody sputum. After another venesection he seemed to be somewhat improved. Several hours after that he suddenly died. The heart had been doing fairly well until the end. He had had digitalis every day. There were no peripheral signs, no edema. There may well have been pulmonary congestion, but probably no extension of his bronchopneumonia. Laboratory studies showed a negative blood culture. The sputum by mouse test showed a few pneumococci. A Hinton test was negative. The urine was negative except that it showed a considerable amount of bile. The white cell count ranged from 29,000 to 34,000 and back to 26,000 four days before death. The chart showed a rapid rise in temperature to 104° until the third day, then coming down, then rising to 101.5° the day of death. The blood pressure had risen to 165/68, then dropped to 112/60. There was a rise of pulse to 150 near the end.

DR. WILLIAM B. BREED: Were there any joint symptoms?

DR. LUDWIG: There were no joint symptoms while he was in the hospital.

DR. AUBREY O. HAMPTON: This film taken nine days before death shows dullness at both bases which on the right is denser and confluent and obscures the outline of the diaphragm, but

these films do not show the typical peeling off of the pleura which we get with fluid. On the right side there are a few round areas of dullness which certainly look like consolidation. The heart is enlarged more downward and to the left than in any other diameter. We see the aortic arch much better than is usual in mitral disease alone.

Clinical Discussion

In Third Year Class

NOTES ON THE HISTORY

Dr. Richard C. Cabot: From the chief complaints we expect that we are going to deal with acute infection. Nothing else is apt to begin that way. We begin to think of the acute infections that are likely to strike a boy of eighteen.

Is an attack that lasts most of the summer "acute"?* Well; here that word is used loosely. I think most people would call it "acute" because it is rheumatism. In other words they use the term to cover cases which last months and even I should say years, with ups and downs. We ought to distinguish this fever from the changes of the bony and ligamentous structures that are sometimes called "chronic rheumatism," having of course nothing to do with rheumatism. This means something which is called "acute" associated with Aschoff bodies and with arthritis, chorea and heart disease.

His father had heart disease. It is generally believed that "acute" rheumatism runs in families.

Very properly the word "grippe" is enclosed in quotation marks. Most diagnoses of grippe are perfectly worthless. I myself cannot speak with authority as to what anybody now thinks about the influenza bacillus. Very few people making a diagnosis of influenza have any knowledge about the influenza bacillus. They make it on a febrile attack, usually with respiratory symptoms, sometimes on gastro-intestinal symptoms, sometimes with no symptoms but fever. In other words a diagnosis of grippe means a short infection of unknown origin. When it is put down as it is put down here, you and I have no idea what it was.

The severe night sweats do not help us at all as to diagnosis. They might mean tuberculosis, but they can occur with all sorts of other diseases. They have no diagnostic value.

Let us sum up before we start on the physical examination. We have a boy who has a well documented history of what sounds very much like rheumatic fever. His father is said to have had heart disease which may have been this kind or some other kind, so that it does not make much difference. Since his seventh year the boy has been short of breath on exertion. While that might not mean anything in a fat old woman, it means a good deal in a thin young man. Rheumatic heart disease therefore is in our minds. Then he has a febrile attack. That febrile attack of course may be due to infarcts or to a fresh rheumatic infection. It may be some respiratory infection quite separate. So we have two things in mind, an old rheumatic infection with valvular disease and very possibly some complicating infection of which we have no further knowledge except that it seems to be respiratory.

*In the summary used by Dr. Cabot there was a note of "an attack of acute rheumatic fever which laid him up for most of the summer".

NOTES ON THE PHYSICAL EXAMINATION

Acute respiratory distress may be cardiac, pneumonic or both. Sweating does not help us at all. Both the things we chiefly think of, heart disease and pneumonia, would give cyanosis.

If he had not had heart disease and if those same physical signs had been told us of the lung, we should have been a good deal surer they meant solidification of the lung than under these conditions. When we have a patient who we have reason to suppose has heart disease, we have also reason to believe that he has congestion of the liver. Then we get pulmonary signs, especially at the right base, much like pneumonia, which postmortem examination shows not to be pneumonia. Therefore be a little cautious of signs like these of solidification at the base in the presence of heart disease. Experience has shown me over the many years I have been following these cases that such signs often mean nothing but a big liver. So we have to bear it in mind as a possibility that all that is written down here might mean nothing but a big liver. How big a liver brings pulmonary signs it is not easy to say. They talk about relaxation of the lung above the liver. That may mean something. The fact is we often get signs like those here given when postmortem shows nothing in the lungs.

The transmission of murmurs* teaches us very little. The textbooks make a great deal more of it than postmortem experience warrants. If a murmur is widely transmitted it means a loud murmur and that is all it means. Any loud murmur is widely transmitted. I have heard murmurs on the top of a man's head; I have heard murmurs—two or three of them—in congenital heart disease without getting nearer than two feet to the patient's chest. If a murmur is loud that is all we need to say about it. Nobody thinks the loudness of a murmur is of much importance in diagnosis. The fact that it goes to the neck is nothing. Any murmur that is loud goes to the neck. They go down too. They may go down to the liver. "Trans-

*In Dr. Cabot's summary it was noted that the systolic murmur at the aortic area was transmitted to the neck.

mitted to the neck'' is one of the stock phrases (there are plenty of them) which we write because we want to write the sort of things other people write. Many of them mean nothing. A systolic murmur and a systolic thrill at the second right interspace, however, are very important diagnostic evidence which we will sum up in a minute after we get the rest of it.

I should like to guess what they mean by ''no definite apical murmurs''. I can guess they mean that they heard something there but it was not so loud as the murmurs they heard elsewhere, so they guessed, perfectly rightly I think, that it was transmitted. Again let me caution you, do not put remarks about ''indefinite''. A murmur is either there or it is not. An ''indefinite murmur'' does not mean anything.

The fact that the aortic second sound was absent is a very important point. As we come to sum up this evidence that is one of the cardinal points. If an aortic second sound is present or loud it does not help you much. If it is absent. that is the writing on the wall; the diagnosis is handed you on a plate. Watch for that. People are always writing down that the aortic second sound is accentuated. It rarely does any good to know that. If it is absent that is a very important thing. With an accentuated pulmonic second sound, as here, it is even more important. The aortic second was not inaudible merely because there was such a noise that they could not hear anything. Some heart murmurs are so noisy that we cannot hear any sounds. Here they got an accentuated pulmonic second sound, in contrast with an absent aortic second sound.

What is the notable thing about that blood pressure?

STUDENTS: The big pulse pressure.

DR. CABOT: Yes. Here is a boy with a good rheumatic history who has had joint symptoms, a characteristic type of disease which ordinarily leads to rheumatic heart disease. He comes in with a very big heart, with a rapid pulse, with one outstanding systolic murmur associated with a thrill in the second right interspace. He also has a loud diastolic murmur which is said to be basal. That is associated with Corrigan pulse and pistol shot sound. The aortic second sound is absent. We have nothing to make us suppose he had a low pitched rolling murmur at the apex; therefore we have nothing definitely to incriminate the mitral valve. But the diagnosis on that valve is a matter partly of inference; I will come back to it in a moment. In regard to that aortic murmur we have reason to suppose two things, stenosis and regurgitation. I do not care a great deal ordinarily whether the pathologist altogether backs me up on these or not. I think the evidence we have here is as good as any evidence on this particular point. If Dr. Mallory says there is no disease in the aortic valve I will retire. If he were to say there was stenosis and no regurgitation or regurgitation and no stenosis I should greatly differ. I think the clinical evidence on that is sufficient. I am not going to submit altogether to the pathologist on that point. Ninety-nine times out of a hundred we must submit. I am going to say aortic stenosis and regurgitation, of the rheumatic type I suppose. I have not gone into the question of type. It might be subacute bacterial endocarditis. We shall come back to that. We have a lesion of the aortic valve. We have no evidence against the mitral valve. But we cannot say there is no mitral trouble. On the evidence before us there is aortic trouble. Very possibly there is mitral trouble too, because it is so common in the rheumatism of children.

The finding of 28,850 white cells is not of any great importance. Almost any of the types of heart disease we think of would produce that. What is more important is 80 per cent polynuclears, hemoglobin 70 per cent, reds 5.3 million. My business is to get the facts before you and to indicate the reasoning that any impartial man would be compelled to follow. In over thirty years' experience in this hospital one of the things I have learned is that 70 per cent hemoglobin rarely means anything. The house officer puts that down under all sorts of conditions. I think he often finds 50 or 40 and 80 or 90; I do not believe he often finds 70. It is a safe reading if you are in a hurry and do not feel sure of yourself. Nobody can say you made a terrible mistake. But I think accurate readings of the hemoglobin very seldom read 70 per cent. The only importance of that here is in its bearing on the differential diagnosis between acute rheumatism and subacute bacterial endocarditis. The latter produces anemia in half or more than half of the cases, while rheumatism very rarely produces anemia. If you are in doubt as to prognosis between a disease that always kills and a disease that gets well, anemia is important as a guide. Therefore the hemoglobin is very important here. I notice that the smear is normal. Does that have any bearing? Could it be normal if we have 5.3 million reds and 70 per cent hemoglobin? But it was normal. Hemoglobin is a hard thing to measure; a smear is easy. Achromia is a thing that jumps right at you. Therefore I believe this blood is normal.

The chart shows a rise in temperature to 104°; then it jogged along not far above normal. He was here nine days. In that time the pulse was gradually getting down. The chart looks as though he was getting better. The respiration got down to normal. They did not think this man was going to die. Something must have come up quite unexpectedly.

He had very good treatment it seems to me.

From that chart it looks as if they came near to saving his life.

It is a very important thing to get more and more evidence about the value of the oxygen tent not only in pneumonia, where we use it so much, but in these heart cases also. It certainly helped him.

Of course the phrase "mitral diastolic and systolic murmurs were well heard" is a diagnosis, not a record of physical signs. What we learn is that the man that wrote that down believed that the murmurs were due to mitral disease. That means he heard a presystolic and a middiastolic of low pitch at the mitral orifice.

DR. WILLIAM C. MARTIN: These are portable films. I do not think they will help much except to show that there was definite disease at both bases.

DR. CABOT: Would you like to prophesy whether that is congestion or pneumonia?

DR. MARTIN: No.

DR. CABOT: That is what we want to know. Do you think it might be nothing but the effects of heart disease without infection?

DR. MARTIN: When we see a large heart like that we usually think such a lung picture as that is probably due to the heart.

DR. CABOT: Not to pneumonia?

DR. MARTIN: This is a portable film.

DR. CABOT: The evidence so far as we have it is against pneumonia, but this being a portable film the radiologist does not want to put the full weight of his authority on that side. I should agree with Dr. Martin that when we know a person has heart disease and the signs are chiefly at the right base it is better not to say pneumonia.

When the oxygen ran out they got the sort of test we should like to make much oftener. It is not humane when a person needs an oxygen tent to take it away and put it on again. But the giving out of the supply of oxygen was very fortunate for our scientific advancement. It seems to me it gives you and me more confidence in the oxygen tent.

Why should a man with heart disease have hemoptysis?

STUDENTS: From passive congestion.

DR. CABOT: If you make a statistical compilation as I have done you will find that passive congestion is the commonest of all causes for raising blood, commoner than tuberculosis. The chief thing to learn is that when a person coughs up blood it may not be due to tuberculosis. Hemoptysis is what we expect in anybody who has a great deal of back pressure on the lung. We see it especially in mitral disease and not in aortic. If you think this is pure aortic disease it should rather surprise you that there is so much pulmonary congestion.

DIFFERENTIAL DIAGNOSIS

I have already committed myself to a diagnosis of aortic stenosis and regurgitation of rheumatic origin. Later in the record you remember they committed themselves to a diagnosis of mitral disease, and I do not know any reason to doubt it. We had better say if a good man thought so, it probably was so; but I wish they had given us their evidence of mitral disease. It is much commoner for rheumatism to attack two valves, especially these two valves, and the mitral much oftener than the aortic. I will say mitral stenosis and regurgitation, probably. I am very confident that we have aortic stenosis and regurgitation, of which they give us excellent evidence.

The question is what else. This x-ray and all the other evidence, hemoptysis, dyspnea and so on, prove passive congestion of the lungs. That is the second thing we should put down. The third thing is passive congestion of the liver. We always get it in these cases. I believe it is the reason for those signs at the right base. What are the classical physical signs of passive congestion of the liver?

STUDENTS: An enlarged tender liver.

DR. CABOT: That is the whole thing. A great many livers are big and not tender at all. The liver of passive congestion is extremely tender. A favorite expression of patients is "It is as sore as a boil." That means passive congestion of the liver. When there is passive congestion of the lungs and liver, on general principles there is passive congestion of the kidneys. Although the urine gives us no reason to prophesy passive congestion, I think that the kidneys are congested. There is no evidence clinically; but why shouldn't they be, when we know he has such a poor heart? I think Dr. Mallory will say passive congestion of the kidneys.

What else? Shall we find evidence of any acute infection in the heart? If so it might be a recurrent rheumatic infection, a formation of fresh granulations on the heart valves on top of the old process, or it might be subacute bacterial endocarditis. I do not think we have the facts in this case to be so sure about that as about the other diagnostic points. In the first place a person can have acute bacterial endocarditis for months and years and have no very distinct symptoms. The mysterious thing about it is how well people feel with acute bacterial endocarditis. He may therefore have had symptoms which are not presented here, or he may have had no symptoms and yet may have had the disease. I do not believe it. I feel that I may be wrong and that if you vote with me for rheumatism you may be wrong. As to acute rheumatic infection on the valves, the only point in its favor is the leukocytosis. That does not help much, because we get such a leukocytosis in people as ill as this with heart disease—though not very often—with no cause in

particular found. It is often associated with infarctions. We have every reason to suppose this boy has infarcts in the lungs and elsewhere. If in the lungs, where else? In the spleen, liver, kidneys,—it is quite possible he has them there. I think it is proved that fever and leukocytosis may be due to infarcts. We do not need to suppose he has infection of any kind merely because he has leukocytosis. There is no evidence of emboli. I never feel sure of any diagnosis of an acute process on the valves unless there are bacteria in the blood stream. I think we are more likely to be right if we say there is no acute process at all. I should say chronic heart disease with failing compensation, the aortic and mitral valves affected.

What about the tricuspid and pulmonary? All we can say about them is that when young people have had heart disease a good while they often have disease of those valves with no physical signs that one can find. Just on pathologic statistics I will say he has tricuspid disease. I do not think any human being can make a diagnosis of tricuspid disease on the facts at hand. It may often be there without physical signs. I want, however, to mention that as one of the probabilities.

A Student: Do you think there is anything in the lungs besides chronic passive congestion?

Dr. Cabot: I think there is some hydrothorax on both sides. I am going to say there is no pneumonia. I realize that pneumonia is a possibility, but if we are going to weigh the evidence as best we can we do not need to say pneumonia.

A Student: He had had dyspnea since he was seven and rheumatic fever when he was fourteen. Is there any chance of a congenital heart?

Dr. Cabot: I should say not. The vast majority of cases of congenital heart disease show themselves before seven. The fact that we have nothing about him before seven is good evidence against it. On the other hand seven is just the time when rheumatic heart disease begins to show itself. Five to fifteen is the favorite age. I believe he very probably had heart trouble from that time.

Dr. Fremont-Smith: Don't you think we have to assume the presence of pulmonary infarcts really to explain the sudden onset of this trouble and the fever?

Dr. Cabot: I think the point Dr. Fremont-Smith brings out is very well taken. The point is, here is a boy with heart disease probably for eleven years. Why does he suddenly get ill and come to the Massachusetts General Hospital? Very likely because after going along fairly well he suddenly began to throw infarcts. They give fever and increase the other symptoms, and it is very possible that the increase of these brought him to his death. On the last day I do not think there is evidence of pulmonary embolism.

Clinical Discussion

At Staff Conference

Dr. Aubrey O. Hampton: The appearance of the heart would go with diffuse dilatation or with rheumatic mitral and aortic lesions. I think that from that picture we would be inclined towards a predominating rheumatic aortic valve lesion with some fluid at the right base and consolidation at both bases. These round areas may be infarcts in the lung.

Dr. Ludwig: At the age of seven the patient had severe heart trouble, probably acute endocarditis.

We thought at admission that he had rheumatic heart disease with mitral and aortic stenosis and regurgitation and bronchopneumonia.

He died of what we thought might be a pulmonary embolus.

Dr. William B. Breed: I do not see any evidence in the record here indicating an embolic death. We know that he had rheumatic heart disease, aortic regurgitation and possibly some mitral stenosis, and pneumonia. I do not see why we have to make a diagnosis of pulmonary infarction in this case.

Dr. Paul D. White: What was the blood pressure?

Dr. Breed: Most of the time the systolic varied from 130 to 150, the diastolic from 40 to 60. He had a high pulse pressure most of the time.

Dr. White: I do not remember from the record whether he had more than one attack of acute dyspnea. Any such acute dyspnea, however, would suggest that he had acute cardiac failure with or without pneumonia or sepsis. With the large pulse pressure and other data one would rather expect that the aortic valve lesion was of growing importance in this case and of greater importance than the mitral lesion.

Clinical diagnoses (from hospital record)

Rheumatic heart disease with mitral and aortic stenosis and regurgitation.
Bronchopneumonia.
Pulmonary infarction.

Dr. Richard C. Cabot's diagnoses

Chronic rheumatic heart disease; aortic and mitral stenosis and regurgitation, probably tricuspid disease.
Hypertrophy and dilatation of the heart.
Failing compensation.
Passive congestion of the lungs, liver and kidneys.

Infarcts in the lungs, spleen, liver and kidneys.
Hydrothorax, bilateral.

ANATOMIC DIAGNOSES

Rheumatic heart disease.
Chronic rheumatic endocarditis of the mitral, aortic and tricuspid valves and the aortic vestibule; mitral, aortic and tricuspid stenosis and mitral and aortic insufficiency.
Cardiac hypertrophy and dilatation.
Acute endocarditis of the mitral and pulmonary valves.
Acute myocarditis.
Rheumatic aortitis.
Chronic rheumatic pericarditis.
Hydropericardium.
Hydrothorax.
Bronchopneumonia, left upper lobe.
Pulmonary edema.
Bronchiolitis, organizing.
Chronic pleuritis, right.
Perisplenitis.
Slight arteriosclerosis, aortic, renal and pulmonary.

Pathologic Discussion

Dr. Tracy B. Mallory: This case is somewhat more interesting anatomically and histologically than clinically. We found a very marked degree of chronic rheumatic endocarditis of the aortic and mitral valves, not quite so severe a chronic endocarditis of the tricuspid valve and acute endocarditis of all four valves. That distribution is typical of the usual progression of the disease. The endocarditis was not limited to the valves. There was definite mural endocarditis in the aortic vestibule. The lesions were somewhat unusual in that the point of maximum involvement seemed to be that cusp of the aortic valve which backs up against the base of the long flap of the mitral valve. There fibrosis had been so extensive that the aortic cusp had been actually pulled off the wall of the aorta. Ordinarily as we open the heart the cusps of the aortic valve cover the sinus of Valsalva. We cannot see the mouth of the coronary artery until the flap of the valve is pulled forward. In this case the posterior cusp of the aortic valve had been pulled down so far that the mouth of the coronary artery was one centimeter and a half above the upper margin of the valve. The valve was practically inside the ventricle, rather than being in the aortic ring. There was also quite extensive endocardial thickening of the entire aortic vestibule running a considerable way down the ventricular wall. It is a very common thing in mitral disease to have the endocarditis running up onto the auricular wall. Somewhat less commonly in aortic disease one sees it running down to the ventricle as one sees it here in such marked fashion. Another finding of some interest was a sharply localized area of gross scarring and tree barking about two centimeters in diameter just below the arch of the aorta, which microscopically showed considerable destruction of the muscularis and some destruction of the elastic fibrils. In itself it would be almost indistinguishable from luetic aortitis; but here we have an eighteen year old boy in whom that can be completely ruled out. It represents without a doubt a healed stage of rheumatic aortitis.

The lungs were distinctly interesting. They were both much larger and heavier than normal, and showed in gross very extensive edema and congestion most of which can be explained as simple chronic passive congestion. In addition however there were some rather small grossly grayish nodules somewhat suggestive of pneumonia, but not entirely characteristic. They seemed unusually firm. We were interested in the possibility of the so-called rheumatic pneumonia that has been described so frequently from New York. When we examined the microscopic sections we found that these nodules consisted of small foci of organizing bronchitis and bronchiolitis. The exact age of the process I think is rather difficult to estimate. I think it must have been going on for at least two weeks, possibly three or four. Interestingly enough we have had one other case of death during the acute stage of rheumatic fever with an exactly identical lesion. Whether it is related to the rheumatic fever or only a coincidence I do not know. In the true rheumatic pneumonitis one is supposed to find lesions in the small pulmonary vessels with secondary hemorrhage into the alveoli. The vessels here seem to be wholly free from acute lesions, and show no more arteriosclerosis than the average case of severe mitral and tricuspid disease. On the other hand a high proportion of the bronchioles show an organizing bronchiolitis, histologically like the organizing pneumonias one sees when pneumonia fails to resolve, except for its localization in the bronchioles rather than in the alveoli.

Throughout the cardiac muscle we have very large areas of acute myocarditis with degeneration of muscle fibers and marked diffuse mononuclear infiltration. There are many peculiar degenerative changes in the heart muscle cells themselves, including appearances suggestive of amitotic division of nuclei. Such changes, though common in voluntary muscle undergoing degeneration, have not been reported to my knowledge in cardiac muscle. This is the type of myocarditis one frequently sees in diphtheria but rarely in rheumatic fever.

Dr. White: Did the fresh endocarditis resemble that due to rheumatism?

Dr. Mallory: Yes. Although blood cultures

showed a staphylococcus albus, I think such growth should be considered a contamination.

DR. WHITE: When the cusp of the aortic valve was pulled down did it show any displacement or narrowing of the coronary mouth?

DR. MALLORY: There were no such changes.

DR. WHITE: Angina pectoris complicating very rare cases of mitral stenosis in young people has been attributed to retraction of the aortic valve or of one aortic cusp along with the coronary artery mouth in the sinus of Valsalva behind it, the retraction being caused by the shortening of the anterior cusp of the stenosed mitral valve. One such case has been reported.

DR. MALLORY: Certainly in this case there was as extreme retraction of the aortic valve as there possibly could be, and I should suppose that the coronary mouth would have been distorted and narrowed if it ever was to be, but such a condition of the coronary mouth was not found.

DR. WHITE: Coburn of New York reported some cases of very severe rheumatic myocarditis and pneumonitis at a meeting in Boston last winter.

DR. MALLORY: We have been looking very carefully for rheumatic pneumonitis of the type Coburn described, but we have not found it yet.

CASE 18412

ONE OF THE NEW INDUSTRIAL HAZARDS

MEDICAL DEPARTMENT

PRESENTATION OF CASE

DR. EARLE M. CHAPMAN*: This is the case of a twenty-nine year old Italian-American laborer who entered the hospital far too ill to give a history. The chief complaints were cough and dyspnea of a year's duration.

Later we found that three years before admission he had entered an industrial plant and had there been employed to stand over an open mixer in which he was mixing silica powder and soap in order to make a well known scouring powder. Following employment in this industry he had been perfectly well for a year and a half, but during the second summer, between twenty-one and twenty-nine months after entering the industry, he developed a dry irritating cough and some dyspnea. Until the following January these symptoms gradually became worse. In January an x-ray was taken by his doctor, who told him he was incapacitated for further work. From that time until entry he had been in about the same condition. He had to limit his activity and had marked dyspnea and cough which had become productive of mucopurulent sputum. Ten days before entry he had a severe head cold with increased dyspnea, orthopnea and chills.

*Resident on the West Medical service.

The remainder of the history is essentially negative.

When he entered the hospital he was obviously very ill. He was sitting up gasping for breath and markedly cyanotic. His face was flushed. On his back there were fine dry scaling lesions. The chest showed an increased anteroposterior diameter and was slightly asymmetrical. The excursion of the diaphragm was limited. There was dullness above the third ribs anteriorly with increased breath sounds, voice sounds and tactile fremitus. In the posterior region there was marked dullness to flatness, with vocal resonance increased below the third rib. The sounds were almost obscured in some areas by coarse crepitant sibilant and sonorous râles. At the left apex anteriorly we thought that the signs were those of cavitation. The voice was amphoric in quality. At the bases the breath sounds were markedly diminished and could hardly be made out. The remainder of the examination was not remarkable. The spleen and liver were not felt. There was no clubbing of the extremities.

Laboratory examination showed the urine normal. The red blood count was 5,100,000, the hemoglobin 80 per cent. During his course in the hospital he ran a persistent leukocytosis from 10,100 to 18,000. 5 per cent eosinophiles was the only remarkable finding in the differential. The stool was not remarkable. Sputum examination showed no tubercle bacilli in four smears. Two examinations showed blood. One specimen was injected into a guinea pig. Its autopsy showed no evidence of tuberculosis.

While he was in the hospital he was very ill and for four or five days we thought he was going to die any moment. Suddenly on the fifth day the crisis occurred. The temperature and the respiratory rate dropped very suddenly. This of course was while he was in an oxygen tent. Beyond this period he still needed oxygen to prevent distress. By the fifth day he was able to remain out of the tent for an hour or two in the morning and afternoon. During one of those periods we found his vital capacity was 350 cubic centimeters, markedly diminished. In spite of that he again developed fever, respiratory distress and tachycardia. Eighteen days after entry he died.

DR. AUBREY O. HAMPTON: I am sorry we have only portable films. The change in the appearance of the chest is very remarkable. Both lungs are grossly abnormal. There is mottled and homogeneous dullness almost completely obliterating the middle four fifths of both lungs and obscuring the outline of the mediastinum and ribs. What appears to be the diaphragm is in about the usual position. There is only a small amount of aerated lung just above both diaphragms and along the left border of the heart and both apices. The trachea is mark-

edly displaced towards the right. The character of the dullness is that of consolidation and incomplete aeration.

Clinical Discussion

Dr. Hampton: We had never seen silicosis that appeared just like this. The dullness you can see is due to complete obliteration of air in the middle four fifths of both lung fields. They are symmetrical and yet at the bases, the apices and along the borders of both sides of the heart he has a very radiant lung.

Dr. Chapman: The admission diagnosis was dyspnea, cyanosis and silicosis. While he was in the ward the diagnosis was acute silicosis with bronchopneumonia.

Dr. J. H. Means: I saw this patient, followed him day to day. I think I have nothing to add to what Dr. Chapman has given. It is a very extraordinary situation.

It may be worth while to mention a report of Dr. Eugene Kilgore at Atlantic City on five cases similar to this one. Their acute course was very similar to the one Dr. Chapman has described to this case. Only one of Dr. Kilgore's patients died. Dr. Kilgore's patients worked in the polish industry, a polish of some sort containing silica dust. The symptoms were all the same. Kilgore mentioned substernal oppression as a characteristic feature of his cases. It seems clear that the polish type of silicosis is similar to the sand soap type.

I wish that Dr. Chapman would say a word about other cases in this industry and emphasize the difference between the acute and chronic types of silicosis, for he has looked up the subject in the literature. He found one English paper.

Dr. Chapman: The first note I saw of these cases was last summer. MacDonald[1] reported in England the cases of two young girls employed in a similar industry as packers. They had inhaled a silica soap dust while packing it and died, one after two and three-quarters years of employment, the other after four and one-quarter years. In Barre, Vermont, in the stone industry Russell[2] reported that he found only the earliest signs by x-ray after two years' exposure, and rarely do symptoms come as early as that; in fact the process in stone cutters is very slow. In cases reported by Britton[3] in which men were exposed in an industry the symptoms of silicosis have developed as long as thirteen years after leaving the industry. So it is probable that the disease was very slowly advancing. We are still looking for an explanation of the more rapid cases that occurred in England, but we have the idea that perhaps the rapidity of the process is due to the fact that soap mixture is alkaline. In the ordinary body tissue the formation of silica hydrosol is a very slow one; when we have alkali present it hastens the process. Apparently that is what happens here.

In support of this theory we tried to determine the actual amount of silica present in the lungs. So after autopsy in this case we did silica determinations and found that there was only about 12 to 13 milligrams of silica present in one gram of tissue, whereas in chronic cases reported before, the quantity of silica was very much higher than that, 100 to 300 milligrams. These data suggest that the reaction in the lungs is not a direct, quantitative one but due to these chemical changes.

Clinical Diagnoses (From Hospital Record)

Silicosis, acute.
Pneumonia.

Anatomic Diagnoses

Acute silicosis.
Silicosis of the bronchial, mediastinal and retroperitoneal glands.
Compensatory emphysema.
Acute and chronic pleurisy.
Bronchopneumonia, slight.
Bronchitis.
Hypertrophy of the right heart.
Arteriosclerosis.
Right hydrocele.
Accessory spleen.
Hepatitis.
Splenitis.
Beginning fibrosis of the bone marrow.
Hyperplasia of the bone marrow.

Pathologic Discussion

Dr. Tracy B. Mallory: The lungs, as you can see from the specimen and the pictures, present an extraordinary appearance. Pale raised emphysematous blebs stand up very sharply from the surrounding depressed, darker purplish tissue. The surface of the lungs very closely resembles the configuration of the hepar lobatum of syphilis. Although fairly diffuse, the process seemed most marked in the right middle lobe and the lower part of the two upper lobes. The lower lobes were relatively less involved. The apices of both upper lobes were shrunken so completely that the actual apex of the lung is formed by the lower lobe rather than by the upper lobe. The upper portion of each lower lobe was enormously distended by emphysema.

In the rest of the body there was comparatively little to find except for involvement of the bronchial lymph nodes and a certain amount of extension of the process to some of the retroperitoneal lymph nodes.

Microscopically we find in the pulmonary tissue localized nodules of very acellular fibrous tissue. Their general size and focal character is not essentially different from that of nearly healed tubercles. These foci often have a con-

glomerate character as tubercles do. The peripheral portions show lymphocytes and mononuclears, but the actual central portion of the lesion is almost completely acellular. A few little refractile granules can be seen here and there which are probably silicon dust. In this type of disease visible deposits of it are not striking. In certain similar cases described as asbestosis, occurring in people working in asbestos, we get extraordinary particles of asbestos in the lungs which particles have a corkscrew appearance and somewhat suggest giant treponemata.

The histologic character of the lesion is such an apparently very chronic one that it is hard to believe the clinical course could be so rapid. It does not seem possible to explain the progression on a histologic basis.

DR. HAMPTON: Didn't you have two other cases from the same place?

DR. CHAPMAN: This man told us of two other cases, one in a foreman, the other in a second man who was employed under him[4]. Of these one is dead and one living. The last time I saw the living patient he told me he had found two other possible cases of the disease in men who had similar symptoms and were incapacitated after doing the same work, and also knew of two girls in the plant who were ill but were afraid to go to a doctor. The foreman also remembered three more men formerly employed in this plant who afterwards died very suddenly of what was called pneumonia. In all likelihood there have been ten cases or more from this one plant.

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1 MacDonald, G., Piggot, A. P., and Gilder, F. W.: Two cases of acute silicosis. Lancet 2:846 (Oct. 18) 1930.
2 Russell, A. E., Britten, R. H., Thompson, L. R., and Bloomfield, J. J.: The health of workers in dusty trades. U. S. Pub. Health Bull. 187, July 1929.
3 Britton, J. A., and Head, J. R.: Pneumoconiosis. J. A. M. A. 96:1938 (June 6) 1931.
4 Chapman, Earle M.: Acute silicosis. (See Case 2 and Case 3.) J. A. M. A. 98:1437 (April 23) 1932.

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RURAL HEALTH

THE current issue of *The Commonhealth*, the quarterly bulletin of the Massachusetts Department of Public Health, is devoted to a critical analysis of the problem of rural health, comprising a symposium by eighteen authorities on various phases of the subject. As Dr. Bigelow writes in his foreword, "It used to be that the morbidity and mortality rates of the country were better than those of the early unsanitary cities. But not so today since our knowledge has been put to work. . . . Perhaps the most obvious difficulty is economic. The delivery of a given standard of quality of service in any of the fields just mentioned to a given unit of the population costs more per unit the more sparsely settled are the people served."

The first county health unit in the United States was established as the result of an epidemic of typhoid fever in Yakima County, Washington, in 1911. Meanwhile, according to Dr. Wilson G. Smillie, writing on the development of full-time county health service in the United States, the Rockefeller Sanitary Commission had been conducting a campaign for the control of hookworm disease in the southern states. The results of this campaign were found to be most satisfactory when a small, permanent nucleus with a continuous comprehensive program of gradual sanitary betterment remained in the area. Temporary grants were accordingly made toward the development of county health units with full-time personnel. From the first the method proved effective; in 1918 there were some thirty county health units with full-time personnel in the United States; by 1932 over 500 counties had adopted the principle, and over 25 per cent of the rural population were so served.

The basic personnel, according to the standard plan, consists of a health officer, one or more nurses, a sanitary inspector and a secretary, all on a full-time basis. The standard budget for a minimum personnel is in the neighborhood of $10,000. The basic activities of the health unit are.

1. Collection and analysis of mortality and morbidity statistics.
2. Communicable disease control.
3. Environmental sanitation.
4. Maternal and infant hygiene.
5. Preschool and school hygiene.
6. Health education.

In Massachusetts, where the county is not the important political organization which it is in other parts of the country, voluntary intertown coöperation has been found to produce the same beneficial results. As early as 1888 communities in the state began grouping themselves into districts for the purpose of employing full-time school superintendents. The first definite amalgamation for the purposes of health protection came about in 1920, when eleven towns in Barnstable County joined voluntarily for health work. This was eventually put upon an official county basis, and today exists as the only county health department in New England. Two other health districts, not formed upon county lines, have more recently been organized—the Southern Berkshire District and the Nashoba Health District. The staff of each unit is headed by a full-time medical health officer, with whom is associated a group of full-time subordinate personnel who assist in carrying out the different phases of the local public health program. The basic activities of the district health unit consist of the following:

1. Controlling communicable diseases.
2. Guarding the hygiene of the individual.
3. Promoting sanitation.
4. Assembling records and statistics.
5. Carrying on public health education.

Every effort is made to supplement and not to supplant the family physician.

This outline of the development of rural health in Massachusetts is the contribution of Dr. Wilson W. Knowlton, district health officer of the department. Other articles tell the story of the development and application of the work in the separate units and present the various problems which are encountered in the struggle to put rural health on a parity with urban health.

ENGLISH-SPEAKING STUDENTS OF MEDICINE AT THE UNIVERSITY OF LEYDEN

THE University of Leyden, founded in 1574 as a Protestant university, soon became extremely popular for English-speaking students who wished to study medicine. Its only serious competitor, at the beginning of its career, was the University of Louvain, strictly reserved, as it still is, for Roman Catholics. In the seventeenth century, when Great Britain was distracted by civil war, puritanism combined with economic conditions drove many people to Holland; among them were students, either intent on medicine as a career or dilettanti who took the study of medicine as part of the education of a gentleman. Almost at the start the young university became a famous medical school and its earliest teachers, Forest and Bontius, were well qualified men who had studied at Padua. By 1578 the first medical student was matriculated, for there had already been established an anatomical theatre, a physic garden, and a library. This student, John James, an Englishman, was graduated M.D. in 1581 and became a distinguished physician in London. Thus James began an invasion of the University of Leyden by students from Great Britain, which was to culminate in the great period of the University as a medical school, under Boerhaave, the first teacher of medicine at the bedside, in the early part of the eighteenth century.

The list of these English-speaking students has now been published, the result of a long continued and energetic search by Dr. Innes Smith*. He has carefully gone through the names of all the students who matriculated during the three hundred years from 1575 to 1875, from a list, the *Album Studiorum*, which was published in the latter year. Additional data were found in *An Index to the English Speaking Students who have Graduated at Leyden University*, published by Edward Peacock in 1882. Peacock's book is not entirely satisfactory, for only a minority of medical students who matriculated also graduated at Leyden and moreover, a considerable number never graduated at all, and the majority graduated elsewhere. Dr. Innes Smith's valuable contribution is much more complete, for he has not only traced many of the students to other universities, but he has given a brief account of their careers, or at least a reference to an authentic biography, if such exists. His studies, begun as an amusement during an illness, gradually extended until they became a serious research, for he has examined lists of students in practically every university of importance graduating men in medicine during the seventeenth and eighteenth centuries, seeking further knowledge of his Leyden matriculates. In many cases he has been able to trace, also, the students' theses, which every man was required to defend, either during his course or at the time of his graduation. Some of these, in manuscript before 1600, are only to be found in the library at Leyden. After that date they were printed and Leyden has a complete run from 1655 onwards. Dr. Smith's researches have carried him to the great libraries of the world and he has been able to find many of the missing dissertations at the British Museum, the Bodleian, and elsewhere. Some of the earliest theses printed by the Elzevirs are of the greatest rarity.

The influence of Leyden as a medical school was immense. Students under Boerhaave carried his message throughout the world and literally hundreds of graduates spent at least part of their course under the medical faculty of this great university. The list which Dr. Innes Smith has so carefully prepared contains the names of many students from England, Scotland and Ireland, but not a few from America. Our first physician, Laurentius Bohun, doctor to the Colony of Virginia, who came to America in 1610, received his M.D. degree at Leyden in 1603. About twenty young men from America went to Leyden during the three hundred years for their degrees: Benjamin Waterhouse (1780), John Redman (1748) of Philadelphia, Roger Price (1719), later rector of King's Chapel, Boston, and others of lesser note.

*R. W. Innes Smith, M.D. English-Speaking Students of Medicine at the University of Leyden. Oliver & Boyd, Edinburgh & London, 1932. xxiii + 258 pages.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

HESS, ALFRED F. B.A., M.D. Columbia University College of Physicians and Surgeons 1901. Clinical Professor of Pediatrics, University and Bellevue Hospital. His subject is "Diet, Nutrition and Infection". Page 637. Address: 16 West 86th St., New York, N. Y.

THORNDIKE, AUGUSTUS JR. M.D. Harvard University Medical School 1921. F.A.C.S. Surgeon in the Department of Hygiene, Harvard University. Assistant in Surgery, Harvard Medical School. Assistant Surgeon, Children's Hospital and Milton Hospital, Milton. His subject is "Acute Recurrent Intussusception in Children." Page 649. Address: 319 Longwood Ave., Boston.

HILL, LEWIS WEBB. A.B., M.D. Harvard University Medical School 1913. Associate Physician, Children's Hospital. Instructor in Pediatrics, Harvard Medical School. His subject is "The Infantile Eczema Problem." Page 655. Address: 319 Longwood Ave., Boston.

BALCH, FRANKLIN G. JR. A.B., M.D. Harvard University Medical School 1923. Assistant Surgeon to Out-Patients, Massachusetts General Hospital, and Faulkner Hospital. Assistant in Surgery, Harvard Medical School. His subject is "Pulmonary Embolism Following Simple Fracture." Page 660. Address: 279 Clarendon St., Boston.

DOWNING, JOHN GODWIN. A.B., M.D. Harvard University Medical School 1915. Assistant Dermatologist, Boston City Hospital and Beth Israel Hospital. Teaching Assistant Dermatologist, Harvard Medical School. Instructor in Dermatology, Tufts College Medical School. Dermatologist, St. Elizabeth's Hospital. His subject is "Pigmentation From Perfume. 'Berlock' Dermatitis." Page 660. Address: 520 Commonwealth Ave., Boston.

HORNING, BENJAMIN G. M.S., C.P.H., M.D. Harvard University Medical School 1928. Epidemiologist, Connecticut State Department of Health. His subject is "Smallpox in Utero." Page 663. Address: 165 Capitol Avenue, Hartford, Connecticut.

PUTNAM, TRACY J. M.D. Harvard University Medical School 1920. Instructor in Neuropathology, Harvard Medical School. Assistant Visiting Surgeon for Neurosurgery, Boston City Hospital. His subject is "Advances in Neurological Surgery." Page 664. Address: Boston City Hospital, Boston.

AN APPEAL FOR ACTION

THE COMMITTEE ON STATE AND NATIONAL LEGISLATION OF THE MASSACHUSETTS MEDICAL SOCIETY

8 The Fenway, Boston.

Dear Doctor:

The Initiative Petition of the Chiropractors will appear on the official ballot this November and the voters will be asked to favor or oppose it.

There are twenty-five "Drugless Healers" to every one hundred graduates of recognized medical schools in the United States. According to an Editorial in the *Boston Herald* of June 30, 1932, five hundred and thirty-six Chiropractors were licensed in one year in New Jersey.

For the past ten years all of their bills have been defeated by legislative action in Massachusetts and in 1932 they were given leave to withdraw by a vote of 151 to 50 in the House and 21 to 13 in the Senate.

Governor Ely defeated the then Governor Allen by a vote of over 606,000 to about 590,000 in the 1930 election. We number about 4800 members. If each physician will influence one hundred votes, we will defeat the petition.

You are earnestly requested to solicit the votes of your friends, patients, those with whom you trade, and particularly your pharmacists, hospital associates and employees.

There should be but one standard for fitness to practice the healing art in Massachusetts, namely, passing the examination of the Board of Registration in Medicine.

Sincerely yours,

H. G. STETSON, *Chairman,*
T. J. O'BRIEN,
F. E. JONES,
A. W. MARSH,
SHIELDS WARREN, *Secretary-Treasurer.*

MISCELLANY

UNIVERSITY EXTENSION CLASSES

Among the variety of instructive courses which have been arranged by the Department of Education, attention should be called to those which have definite relation to medicine and hygiene.

One under the title "Human Nature and Society" will be conducted at the Old South Meeting House, Washington and Milk Streets, Boston. The first lecture will take place on Wednesday, October 19, from 7:45 to 9:30 P.M. and will be followed by seven additional lectures of one semester hour each. The charge for this course is $4 for auditors and $5 for credit students, with textbooks extra. The instructor is Dr. Abraham Myerson, Professor of Neurology and Neuropathology, Tufts College Medical School.

Another course, under the title of "Normal Youth and Its Everyday Problems," will be given in Room 2, Harvard Hall, Harvard University, Cambridge. The first lecture is scheduled for Monday, October 24, from 7:30 to 9:15 P.M. This is to be followed by seven additional lectures, the charge for this course being $4 for auditors and $5 for credit students, with text materials extra. The instructors are Dr. Douglas A. Thom, Director, Division of Mental Hygiene, Massachusetts Department of Mental Diseases; Dr. Samuel W. Hartwell, Director, Worcester Child Guidance Clinic and Dr. Henry B. Elkind, Medical Director of the Massachusetts Society for Mental Hygiene. Note: As this course deals with the psychology of adolescence, it may be offered as the required course in that subject by graduates of State Teachers' Colleges who are completing their work for a degree through University Extension courses.

"Keeping Mentally Fit" is another subject which will be dealt with at Harvard University. "Psychology Applied to Business, Personal and Social

Problems" is scheduled for Room 6, Harvard Hall, Harvard University, Cambridge, on Monday, October 31, from 7:30 to 9:15 P.M. Seven additional lectures of one semester hour each will follow. The charge is $3 for auditors and $5 for credit students, with textbook extra. This course will be followed by one on the "Psychology of Self-Development."

"Social Problems (Applied Social Psychology)" will be considered in the Gardner Auditorium, State House, Boston. The first lecture is to be given on Thursday, November 3, from 7:45 to 9:30 P.M. This course will consist of sixteen lectures of two semester hours each. The charge will be $5 for one part of eight lectures or $8 for the complete course. The instructor is Walter Ballou Jacobs, Professor Emeritus of Brown University. In this course teachers, students, nurses, social workers, professional and business men and women and parents will be especially instructed.

The "Psychology of Personality" will be dealt with in the Gardner Auditorium, State House, Boston. The first lecture is scheduled for Tuesday, November 1, from 7:45 to 9:30 P.M. There will be eight lectures in this course with the charge of $3 for auditors and $5 for credit students, with textbook extra. In this course the elements that make up one's personality are studied and practical measures are suggested for improving elements that are defective.

"How to Keep Healthy" will be considered in Room 5, Harvard Hall, Harvard University, Cambridge. The first lecture is scheduled for Monday, October 24, from 7:30 to 9:15 P.M. This course is offered in two parts of eight lectures each; two semester hours. The charge for each part is $3 for auditors, $5 for credit students; textbook extra. The lecturers are Dr. Milo Green, Boston University School of Medicine, Dr. Lloyd T. Brown, Harvard Medical School and others. The course leader will be Dr. Nathan H. Garrick, Professor of Neurology and Director of Health Service, Boston University.

"Health Education" will be considered in Room 2, Harvard Hall, Harvard University, Cambridge. The first lecture is scheduled for Thursday, October 27, from 7:30 to 9:15 P.M. There will be fifteen lectures in this course of two semester hours each. The charge will be $6 for one part of eight lectures, $10 for the complete course. The instructor is Clair E. Turner, Professor of Biology and Public Health of the Massachusetts Institute of Technology.

CORRESPONDENCE

STERILIZATION OF LIGATURES

October 6, 1932.

The *New England Journal of Medicine*
8 The Fenway
Boston, Mass.

To the Editor

Dear Sir:

As manufacturers of ligatures and sutures for surgical purposes, we are naturally interested in your editorial "Sterile Surgical Catgut," page 375, August 25, 1932.

We have never released a tube of catgut until samples of its lot were reported sterile by a competent bacteriologist. The Sias Laboratories in the Brooks Hospital have done this testing according to the method recommended by Meleney and Chatfield since its publication in 1931.

Yours very truly,
E. F. MAHADY COMPANY.

NOTICES

CLINICS IN MEDICINE AT PETER BENT BRIGHAM HOSPITAL

On Thursdays at 3:30 P.M. in the Amphitheatre of the Peter Bent Brigham Hospital Dr. Henry A. Christian, Hersey Professor of the Theory and Practice of Physic, Harvard, and Physician-in-Chief, Peter Bent Brigham Hospital, will give a series of clinics which members of the medical profession are cordially invited to attend. The next clinic will be given on October 20.

Also physicians and medical students are invited to attend ward rounds of the Medical Staff of the Peter Bent Brigham Hospital held from 10-12 o'clock on Saturdays.

THE SOCIETY OF PLASTIC AND RECONSTRUCTIVE SURGERY

ANNUAL MEETING
OCTOBER 28 AND 29, 1932
NEW YORK CITY

New York Academy of Medicine, New York Hospital and Cornell University Medical College, Sydenham Hospital, Hospital for Joint Diseases, Mount Sinai Hospital, Post-Graduate School and Hospital, New York Skin and Cancer Hospital.

The profession is invited to attend.

PROGRAM

FRIDAY, OCTOBER 28, 1932

Dry Clinics and Round Table Conferences

1. Hospital for Joint Diseases, 1919 Madison Avenue.
   9:00 to 10:30 A.M.—Reconstructive procedures in orthopedic surgery. Presentation of cases. Dr. Leo Mayer, Dr. Harry Finkelstein.
2. Mount Sinai Hospital, 1 East 100th Street,
   11:00 to 12 M.—Plastic repair in ophthalmology. Presentation of cases. Dr. I. Goldstein.
3. Sydenham Hospital, Manhattan Avenue and 124th Street.
   2:00 to 3:30 P.M.—Plastic surgery of face, neck, breast and extremities. Pre- and post-operative. Presentation of cases, Dr. J. W. Maliniak.

Special Session

FRIDAY EVENING, OCTOBER 28, 1932, AT 8:30 P.M.
New York Academy of Medicine
2 East 103rd Street

Friday, October 28, promptly at 7:00 P.M., a din-

ner will be held for members of the society at the New York Academy of Medicine, 2 East 103rd Street, preceding the evening session. *Formal.*

Reservations should be sent to Dr. Gaston Labat, General Secretary, 30 East 40th Street.

I. Reading of the Minutes
II. Election of Active and Corresponding Members
III. Scientific Session

1. Chairman's Address.
   Plastic surgery as an organized specialty and problems confronting it. Dr. J. W. Maliniak.
2. Surgical treatment of cleft palate. Dr. Harold S. Vaughan.
3. Tendon and nerve repair in injuries of the hand. Dr. Leo Mayer.
4. Technical considerations in plastic surgery of the breast. Dr. Claoue (Paris).
   Discussion opened by Dr. Robert Ivy, Philadelphia; Dr. Harry Finkelstein, Dr. Carl G. Burdick.

SATURDAY, OCTOBER 29

Dry Clinics

1. Post-Graduate School and Hospital, 303 East 20th Street.
   9:00 to 10:00 A.M.—Cleft lip and cleft palate, Dr. Vaughan. Oral surgery, Dr. Vaughan.
2. New York Skin and Cancer Hospital, 301 East 19th Street.
   10:30 to 11:30 A.M.—Plastic surgery, Dr. Clarence Straatsma.
3. New York Hospital and Cornell University Medical College, 1st Avenue and 28th Street.
   12 Noon—Lunch will be served.
   12:30 to 1:30—Reconstructive procedures in otolaryngology. Presentation of cases. Dr. Arthur Palmer, Dr. H. Lyons Hunt.

Afternoon Program

Auditorium of New York Hospital and Cornell University Medical College, 1st Avenue and 28th Street.

I. Executive Session
   Report of the Committees
II. Scientific Session
   a. Radium as an adjunct to plastic surgery of the head and neck. Dr. G. Allen Robinson.
   b. The value of osteocartilagenous grafts in rhinoplasty. Dr. W. W. Carter.
   c. The use of fascia in reconstructive surgery. Dr. C. M. Gratz.

   Discussion by Dr. Leon T. LeWald, Dr. Ira Kaplan, Dr. Gustav J. Tieck, Dr. Clarence Straatsma.

REMOVALS

Dr. Allen R. Barrow announces the removal of his office to 19 Bay State Road, Boston.

Dr. Franklin S. Newell announces the removal of his office to 221 Longwood Avenue, Boston.

ANNOUNCEMENT

Dr. Frank L. Newton announces his intention to resume practice in October at Hillcrest, 123 Highland Avenue, Somerville, Massachusetts.

DR. ROBERT T. MONROE BECOMES ASSOCIATED WITH DR. HENRY A. CHRISTIAN

Robert T. Monroe, M.D., University of Michigan, 1924, has become associated with Dr. Henry A. Christian, Physician-in-Chief, Peter Bent Brigham Hospital, in consultation practice of internal medicine with office at the Peter Bent Brigham Hospital, Boston.

REPORTS AND NOTICES OF MEETINGS

ESSEX NORTH DISTRICT MEDICAL SOCIETY

The next meeting of the Essex North District Society, a joint meeting, will be held at Raymond's Sunnyridge Farm, at Amesbury, on Wednesday, October 19, at 1 P.M. Dinner from 1 to 2. Dr. H. G. Stetson, president of the Massachusetts Medical Society, will speak for twenty minutes on matters of current interest in the parent society. Dr. Frank Ober will speak on the general management of arthritis, forty minutes, followed by a general discussion.

The semi-annual meeting will be held on Wednesday, January 4, and the annual meeting on Wednesday, May 3, programs to be announced.

E. S. BAGNALL, M.D., *Secretary-Treasurer.*

SUFFOLK DISTRICT MEDICAL SOCIETY

CENSORS' MEETING

The Censors of the Suffolk District Medical Society will meet for the examination of candidates at the Medical Library, No. 8 The Fenway, Thursday, November 3, 1932, at 4:00 o'clock.

Candidates should make personal application to the Secretary, and present their medical diplomas at least one week before the examination.

LELAND S. MCKITTRICK, M.D., *Secretary.*
205 Beacon Street, Boston.

PROGRAM OF CLINIC FOR THE SUFFOLK DISTRICT MEDICAL SOCIETY AT THE BOSTON CITY HOSPITAL

Wednesday, October 26, 1932, Cheever Amphitheatre, 8:15 to 10 P.M.

Initiative Petition of Chiropractors. Dr. Thomas J. O'Brien.

Certain Atypical Manifestations of Subacute Bacterial Endocarditis. Demonstration. Dr. Soma Weiss.

Cardiac Hypertrophy. Dr. F. B. Mallory.

Treatment of Tetanus. Dr. William R. Morrison.

New Growths of Large Bowel. Dr. Arthur R. Kimpton.

Clinical Demonstration of Cases. Dr. Ralph C. Larrabee.

Comparative Mortality Rates of Acute Appendicitis. Dr. Irving J. Walker.

GREATER BOSTON MEDICAL SOCIETY

A joint meeting of the Greater Boston Medical Society and the Beth Israel Hospital will be held on Tuesday, October 18, 1932, at 8:15 P.M., at the Beth Israel Hospital.

PROGRAM

Investigations of the Research Laboratories of the Beth Israel Hospital

The Use of Decholin as a Clinical Test of the Velocity of Blood Flow—Samuel L. Gargill, M.D.

Studies of the Action of Diuretics—Dorothy Rourke Gilligan, M.S.

The Clinical Effect of Insulin Hypoglycemia on the Circulation—Joseph E. F. Riseman, M.D.

Primary Hypochromic Anemia — William Dameshek, M.D.

The Present Status of Female Sex Hormone Therapy—Henry S. Finkel, M.D.

The Problem of Pulmonary Emphysema — David Davis, M.D.

The Significance of the So-Called Peripheral Signs of Aortic Regurgitation with Particular Reference to Duroziez' Sign—Herrman L. Blumgart, M.D.

BERNARD I. GOLDBERG, M.D., *Secretary.*
483 Beacon Street, Boston, Mass.

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

The regular clinical conference of the New England Hospital for Women and Children will be held at the hospital, Dimock St., Roxbury, on Thursday, October 20 at 8 P.M. The program will be in charge of the neuro-psychiatric department. Dr. Louise Eisenhardt will speak on Tumors of the Brain and Spinal Cord, and Dr. Madelaine R. Brown on Meningo-myeloencephalitis.

ALICE H. BIGELOW, M.D., *Secretary.*

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Ampitheatre (Van Dyke Street entrance), Tuesday evening, October 25, at 8:15 o'clock. Program: Presentation of Cases.

The Reconstruction Surgery of Chronic Arthritis, by Dr. Robert B. Osgood and Dr. Philip D. Wilson.

JOHN HOMANS, M.D., *Secretary.*

THE NEW ENGLAND ROENTGEN RAY SOCIETY

The October meeting will be held in the Thorndike Amphitheatre at the Boston City Hospital, Boston, Mass., Friday, October 21, 1932, at 8:00 P.M.

SCIENTIFIC SESSION

Congenital Malpositions of Colon and Appendix. Dr. D. O. McKee.

Vertebral Epiphysitis. Dr. M. I. Smedal.

Joint Changes in the Pelvic Girdle. Dr. P. E. Tivnan.

Knee Joint Pathology. Dr. A. M. Moloney.

Sclerosing Osteomyelitits. Dr. F. W. O'Brien.

Diagnostic Problems. Dr. Max Ritvo.

Clinical Radiological Studies. Dr. P. F. Butler.

The usual dinner will be held at the Harvard Club.

C. W. BLACKETT, M.D., *President,*
35 Bay State Road, Boston, Mass.
T. R. HEALY, M.D., *Secretary.*
370 Marlborough St., Boston, Mass.

HAMPDEN DISTRICT MEDICAL SOCIETY

The Regular Fall Meeting of the Society will be held at Monson State Hospital on Tuesday, October 18, 1932. The Society will be guests of Supt. Dr. Morgan B. Hodskins.

Lunch at 1:15 P.M. Following lunch clinical cases illustrating various neurological conditions will be shown.

Fellows who wish to inspect the Hospital can do so, either before the luncheon or after the clinical program.

HERVEY L. SMITH, *Secretary.*

COMBINED MEETING OF THE PEDIATRIC SECTION OF THE NEW YORK ACADEMY OF MEDICINE, PHILADELPHIA PEDIATRIC SOCIETY AND NEW ENGLAND PEDIATRIC SOCIETY

SATURDAY, OCTOBER FIFTEENTH, NINETEEN HUNDRED AND THIRTY-TWO, BOSTON, MASSACHUSETTS

MORNING SCHEDULE

Members arriving from New York and Philadelphia should get off the train at Back Bay station and go directly to the Harvard Club, 374 Commonwealth Avenue, where breakfast will be served from 7:30 to 9:00 o'clock.

Harvard Medical School, Amphitheatre D

10:00 A.M. An Interpretation of the Functions of the Sympathetic Nervous System. Dr. Walter B. Cannon.

10:30 A.M. Some Aspects of Secondary Anemia. Dr. George R. Minot.

11:00 A.M. Basal Metabolism Determinations in Children: Their Clinical Significance. Dr. Fritz B. Talbot.

11:30 A.M. What Is Celiac Disease? Dr. John L. Morse.

12:00 M. What Is Nephrosis? Dr. Henry A. Christian.

12:45 P.M. Luncheon at Longwood Towers, Brookline.

AFTERNOON SCHEDULE

Children's Hospital Amphitheatre, Longwood Avenue

3:00 P.M. Vitamin A Deficiency. Dr. Kenneth D. Blackfan.

3:30 P.M. Demonstration of Patients with Endocrine Dysfunction. Dr. Gerald Hoeffel.

4:00 P.M. Considerations on the Use of Protein and Salt in the Treatment of Various Types of Bright's Disease. Dr. Allan M. Butler.

4:20 P.M. Streptococcus Tracheobronchitis. Dr. Lyman Richards.

4:40 P.M. The relation of Anemia of the Newborn to Familial Icterus Gravis and Erythroblastosis. Dr. Louis K. Diamond.

5:30 P.M. Refreshments at Harvard Club. (Aesculapian Room.)

7:30 P.M. Dinner at the Hotel Kenmore, 490 Commonwealth Avenue.
Dr. George H. Bigelow, Health Commissioner of Massachusetts—The Relationship of Public Health Activities to the Practice of Pediatrics.

OFFICERS

New York Academy of Medicine

Chairman: Dr. John Caffey, 50 Haven Avenue, New York, N. Y.

Secretary: Dr. Harry Bakwin, 132 East 71st Street, New York, N. Y.

Philadelphia Pediatric Society

President: Dr. John P. Scott, 2040 Pine Street, Philadelphia, Pa.

Secretary: Dr John D. Donnelly, 115 Bryn Mawr Avenue, Bala-Cynwyd, Pa.

New England Pediatric Society

President: Dr. Lewis Webb Hill, 319 Longwood Avenue, Boston, Mass.

Secretary: Dr. Gerald Hoeffel, 319 Longwood Avenue, Boston, Mass.

SOCIETY MEETINGS
CONGRESSES AND CONFERENCES

October 15—Combined Meeting of the Pediatric Section of the New York Academy of Medicine, Philadelphia Pediatric Society and New England Pediatric Society. See page 683.

October 17-21—Clinical Congress of the American College of Surgeons. See page 1330, issue of June 23.

October 17-28—The 1932 Graduate Fortnight of the New York Academy of Medicine. See page 1375, issue of June 30.

October 18—South End Medical Club. See page 634, issue of October 6.

October 18—Greater Boston Medical Society. See page 683.

October 19—The New England Dermatological Society. See notice on page 557, issue of September 22.

October 19—The New England Physical Therapy Society. First meeting of the season.

October 20—Clinics in Medicine at the Peter Bent Brigham Hospital. See page 681.

October 20—New England Hospital for Women and Children. See page 683.

October 21—The New England Roentgen Ray Society. See page 683.

October 22 and December 7—American Board of Obstetrics and Gynecology. Announcement of Coming Examinations. See notice on page 557, issue of September 22.

October 22-24—Health Education Institute. See notice on page 47, issue of July 7.

October 24-27—American Public Health Association. See notice on page 1321, issue of December 31, 1931.

October 24-28—Inter-state Postgraduate Medical Association of North America. See page 39, issue of January 7.

October 25—Harvard Medical Society. See page 683.

October 28 and 29—The Society of Plastic and Reconstructive Surgery. See page 681.

DISTRICT MEDICAL SOCIETIES

ESSEX NORTH DISTRICT MEDICAL SOCIETY

October 19—See page 682.

January 4—Wednesday, Semi-Annual Meeting. Program to be announced.

May 3—Wednesday, Annual Meeting. Program to be announced.

HAMPDEN DISTRICT MEDICAL SOCIETY

October 18—See page 683.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

The program of the Middlesex East District Medical Society for the coming year is as follows:

November 9 at Winchester.
January 11 at Wakefield.
March 15 at Melrose.
May, at the Meadowbrook Club, Reading.

ALLAN R. CUNNINGHAM, Secretary.

SUFFOLK DISTRICT MEDICAL SOCIETY

November 3—Censors' Meeting. See page 682.

Joint Meetings of the Suffolk District Medical Society and the Boston Medical Library to Be Held Wednesdays at 8:15 P. M.

October 26, 1932—Stated Meeting. Clinical Program, Boston City Hospital. See page 682.

November 30, 1932—Clinical Program, Peter Bent Brigham Hospital.

January 25, 1933—General Meeting in association with the Boston Medical Library. Titles and speakers to be announced later.

March 29, 1933—Clinical Program, Massachusetts General Hospital.

April 26, 1933—Annual Meeting. Election of Officers. Scientific Program—Titles and speakers to be announced later.

The Medical Profession is cordially invited to attend all of these meetings.

WILLIAM H. ROBEY, M.D., President,
LELAND S. McKITTRICK, M.D., Secretary,
HILBERT F. DAY, M.D., Boston Medical Library.

WORCESTER DISTRICT MEDICAL SOCIETY

Schedule of meetings for 1932-1933:

Thursday, November 3—Fall Censors' meeting. See page 636, issue of October 6.

Wednesday, November 9—Dinner and scientific meeting at the Worcester Hahnemann Hospital. Program to be announced later.

Wednesday, December 14—Meeting at the Worcester Chamber of Commerce Auditorium. Paper by Dr. Joseph Meigs of Boston, entitled "Uterine Bleeding."

Wednesday, January 11—Dinner and meeting at the Worcester City Hospital. Paper by Dr. Morris Fishbein, Editor of the Journal of the American Medical Association.

Wednesday, February 8—Dinner and scientific meeting at the Worcester State Hospital. Program to be announced later.

Wednesday, March 8—Dinner and scientific program at the Memorial Hospital, Worcester. Program to be announced later.

Wednesday, April 12—Dinner and scientific meeting at the Rutland State Hospital. Scientific program to be announced later.

Wednesday, May 10—The Annual Meeting. Place and program to be announced later.

ERWIN C. MILLER, Secretary.